A STUDY TO ANALYSE PREOPERATIVE AND POSTOPERATIVE SEVERE PULMONARY HYPERTENSION FOLLOWING MITRAL VALVE REPLACEMENT FOR MITRAL VALVULAR HEART DISEASE.

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CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY TO ANALYSE PREOPERATIVE AND POSTOPERATIVE SEVERE PULMONARY HYPERTENSION FOLLOWING MITRAL VALVE REPLACEMENT FOR MITRAL VALVULAR HEART DISEASE"** is the bonafide original work of **DR. A. ARUNKUMAR** in partial fulfillment of the requirements for M.Ch. Branch-I CARDIO-VASCULAR & THORACIC SURGERY examination of THE TAMIL NADU DR. M.G.R MEDICAL UNIVERSITY to be held in August 2010. The period of post-graduate study and training was from August 2007 to July 2010.

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DECLARATION

I **Dr. A. ARUNKUMAR,** solemnly declare that this dissertation entitled, **"A STUDY TO ANALYSE PREOPERATIVE AND POSTOPERATIVE SEVERE PULMONARY HYPERTENSION FOLLOWING MITRAL VALVE REPLACEMENT FOR MITRAL VALVULAR HEART DISEASE"** is a bonafide work done by me at the Department of Cardio Vascular & Thoracic Surgery, Madras Medical College and Government General Hospital during the period 2007 – 2010 under the guidance and supervision of the Professor and Head of the Department of Cardiothoracic Surgery, Madras Medical College and Government General Hospital, **Prof. S. Manoharan, M.S., M.Ch.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.Ch. Degree (Branch – I) in Cardio-vascular & Thoracic Surgery.**

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Dr. A. ARUNKUMAR

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Contents

INTRODUCTION

Rheumatic Heart disease has been one of the major health problems in developing countries like India. This is an autoimmune disease which occurs in the cardiac tissues due to streptococcal throat infection. Pancarditis and particularly vasculitis contributes the major complication following this disease. Mitral valve constitutes about 50 % of the valve which gets affected due to rheumatic heart disease. Pulmonary Hypertension is said to complicate about 70% of the patients affected by this disease. Pulmonary Hypertension adversely affects the prognosis and course of the disease.

Pulmonary Hypertension in mitral valvular heart disease leads to various adverse outcome following surgical treatment of this condition. In majority of the patients this Pulmonary Hypertension is reversible following surgery. In considerable number of patients however the persistence of Pulmonary Hypertension leads to postoperative problems which may result in death.

Understanding the Pathophysiology of the Pulmonary Hypertension occurring due to this disease and analyzing it before embarking on the treatment option would largely benefit the team involved in the care of suffering patients.

Management of Pulmonary Hypertension before surgery involves investigations and manipulation with certain drugs which may reduce the Pulmonary pressures before proceeding towards surgical correction.

An attempt of a prospective study to analyze the facts is made and the results are tabulated and compared to the national and international views on the same parameter of this common disease.

Pulmonary Hypertension is diagnosed clinically by identifying loud P2 component of 2nd Heart sound, TR Murmur and the accompanying ECG changes

like Right Atrial enlargement, Right Axis deviation and electrical rotation of the Heart.

Pulmonary Hypertension is readily identified by Echocardiography which is also used to classify the pressures into mild, moderate and severe categories. Catheterization studies are not done routinely as echocardiogram has emerged as an effective tool to identify Pulmonary Hypertension and classify accordingly.

In our study which was conducted for a period of 2 years from July 2007 to July 2009 we had recorded data of about 265 patients with Mitral Stenosis or Mitral Regurgitation cases with severe Pulmonary Hypertension. We had investigated them with preoperative ECHO and intraoperative PAP measurement using transducers and followed them with postoperative Echocardiogram for about 6 months.

All patients were well informed about the procedure and appropriate consent had been obtained prior to the valve replacement.

The results obtained from the data collection were tabulated and analyzed using various parameters which may affect the outcome of the patients and were statistically scrutinized using appropriate software.

Review of Literature

REVIEW OF LITERATURE

PULMONARY HYPERTENSION -MECHANISM

Patients with Rheumatic Heart Disease develop Pulmonary Hypertension due to various reasons and prime cause among them will be the retrograde transmission of LA Hypertension which gets transmitted to the Pulmonary arteries. Pulmonary venous pressure also is transmitted to Pulmonary arteries. There is also reactive Pulmonary Arteriolar constriction which can lead to Pulmonary Hypertension. For some unknown reasons there are morphological changes in Pulmonary Vasculature which can lead to the development of Pulmonary Hypertension. Interstitial Oedema which can occur due to elevated LA pressure >20 mmHg can also lead to the development of Pulmonary Hypertension.

Pathophysiology of Pulmonary Hypertension

Elevated LA pressure

⇩

Elevated Pulmonary venous pressure

⇩

Transmitted pulmonary venous hypertension to arteries

⇩

Vasoconstriction of Pulmonary arterioles

⇩

Pulmonary Artery Hypertension

⇩

Right ventricular dilatation

⇩

Tricuspid Regurgitation

⇩

Right Atrial enlargement

⇩

Congestive cardiac failure

The above flow chart explains the Pathophysiology of Pulmonary Hypertension in Mitral Valvular heart disease in both Rheumatic Mitral Stenosis and Regurgitation.

Pulmonary Hypertension greatly influences the natural course of the disease process, treatment response and also the post intervention prognosis.

Wood et al had noted that severe PHT was associated with both moderate MS and severe MS. Fawzy and Reibero pointed out that there is another group of patients with severe MS, yet only a mild increase in PAP. The reasons for not developing PHT in these cases are not clear. No significant relation between LA size and PAP has been noted. There is a view that the increase in LA size would cushion of the sequelae of increase in pressure.

Bahl et al and Krishnamoorthy et al had observed that PHT is an indicator of disease severity in patients with Mitral Stenosis. Chronicity of the disease process as indicated by the presence of severe fibrosis and Atrial Fibrillation are important in the development of reactive PHT

FEATURES INDICATING SEVERITY OF DISEASE IN MITRAL VALVULAR LESIONS COMPLICATED BY PULMONARY HYPERTENSION

- Severe Subvalvular Pathology
- Small Mitral Valve Area
- Higher Transvalvular gradient
- Higher Pulmonary valve resistance
- Higher NYHA symptoms
- Thickened non pliable valves
- Higher Wilkins score
- Higher incidence of Atrial Fibrillation.

DEFINITION:

In medicine, Pulmonary Hypertension (PH or PHT) is an increase in blood pressure in the Pulmonary artery, Pulmonary vein, or Pulmonary capillaries, together known as the Lung Vasculature, leading to shortness of breath, dizziness, fainting, and other symptoms, all of which are exacerbated by exertion. Pulmonary Hypertension can be a severe disease with a markedly decreased exercise tolerance and heart failure. It was first identified by Dr. Ernst von Romberg in 1891

The Venice 2003 revised classification system can be summarized as follows[4]

WHO Group I - Pulmonary Arterial hypertension (PAH)

Idiopathic (IPAH)

Familial (FPAH)

Associated with other diseases (APAH): Collagen Vascular Disease (e.g. Scleroderma), congenital shunts between the systemic and Pulmonary Circulation, portal hypertension, HIV infection, drugs, toxins, or other diseases or disorders

Associated with venous or capillary disease

WHO Group II - Pulmonary Hypertension associated with left heart disease

Atrial or Ventricular disease

Valvular disease (e.g. Mitral Stenosis)

WHO Group III - Pulmonary Hypertension associated with lung diseases and/or Hypoxemia

Chronic Obstructive Pulmonary disease (COPD), Interstitial Lung Disease (ILD)

Sleep-disordered breathing, Alveolar Hypoventilation

Chronic exposure to high altitude

Developmental lung abnormalities

WHO Group IV - Pulmonary Hypertension due to chronic thrombotic and/or embolic disease

Pulmonary Embolism in the proximal or Distal Pulmonary Arteries

Embolization of other matter, such as tumor cells or parasites

WHO Group V - Miscellaneous

This Classification does not include sickle cell disease, Human Herpesvirus 8, also associated with Kaposi's sarcoma, and has been demonstrated in patients with PAH, suggesting that this virus may play a role in its development. Recent studies have been unable to find an association between human Herpesvirus 8 and Idiopathic Pulmonary Arterial Hypertension.

Pulmonary Hypertension – Heath Edwards grading system

Grade	**Microscopic Features**
	Potentially Reversible
I	Hypertrophy of the media of muscular Pulmonary Arteries. Extension of muscle into the wall of Pulmonary Arterioles.
II	Muscle Hypertrophy plus proliferation of intimal cells in arterioles and small muscular arteries.
III	"Muscle hypertrophy plus sub endothelial fibrosis. Eventually, concentric masses of fibrous tissue and reduplicated internal elastic lamina occlude the vascular lumen of arterioles and small muscular arteries. Large elastic arteries show atherosclerosis."

Grade	Microscopic Features
	Usually Irreversible
IV	"Muscle hypertrophy is less apparent; progressive dilatation of small arteries, especially those near vessels with Intimal fibrous occlusion. Plexiform lesions occur."
V	Plexiform and Angiomatoid lesions plus intra-alveolar Hemosiderin-filled macrophages.
VI	Necrotizing Arteritis with thrombosis. Fibrinoid necrosis of the arterial wall with a Transmural infiltrate of Polymorphonuclear leukocytes and Eosinophils.

Usually Pulmonary Hypertension grade 3 and below are reversible and can be taken up for surgery with better results. Pulmonary Hypertension grade 4 and above has significant contribution for postoperative morbidity and mortality.

PATHOLOGICAL FINDINGS:

Pathological changes in lungs in rheumatic MS include prominent vascular and parenchymal changes. Pulmonary veins develop muscular media. Moderate to marked medial hypertrophy occurs in medium sized branches of the Pulmonary arteries. Dilatation lesions and plexiform lesions are rarely seen in rheumatic MS .Tandon et al and Chopra et al has reported such lesion in 4% of the autopsy studies conducted in New Delhi, India. Mubeen et al, recently has reported that Pulmonary vascular changes do not go beyond grade 3 (Heath Edward) criteria in RHD. The most striking feature seen is prominent smooth muscle layer in Bronchoalveolar walls. The extent and severity of both vascular and parenchymal changes are seen more in juvenile MS. Haemosiderosis has been reported in long standing cases.

TREATMENT OPTIONS:

- MEDICAL
- INTERVENTIONAL
- SURGICAL

MEDICAL:

Patients with Mitral Valvular heart disease and PHT are started with rate controlling drugs and diuretics once they become symptomatic. Rate controlling drugs like Beta-blocker and Calcium channel blockers are started to control AF which otherwise would worsen the condition. Rate control with these drugs help in reducing the Transmitral gradient and hence the PHT. Digoxin is usually started for the failing heart and RV dysfunction which has to pump blood against severe Pulmonary Hypertension.

INTERVENTIONAL:

Isolated MS and PHT were treated by closed Mitral Commisurotmy (CMC) and Balloon Mitral Valvotomy (BMV) from time immemorial. Balloon Mitral Valvotomy can be attempted only if the valves are pliable and the Wilkins score is favorable. There are many concerns before proceeding to BMV like the less tolerance to the stress of the procedure, difficulty in negotiating the septum in large right sided chambers, Tight Mitral Stenosis and fear of tearing the mitral valve and creating Mitral Regurgitation. However the AHA GUIDELINES 2008 clearly states the role of BMV in a case of severe PHT associated with rheumatic heart disease. Even though BMV has given good results in MS and PHT the mitral valve area remained less on comparison with open procedures with reasonable reduction in Pulmonary vascular resistance and PAP.

SURGICAL:

Patient's hemodynamic performance was better following reduction in Pulmonary vascular resistance particularly after MVR and valvotomy. Pulmonary Hypertension regresses after the transmitral gradient reduced following surgery. Hemodynamic studies done in patients who had undergone MVR showed a reversibility of Pulmonary Artery Hypertension[19]. Various options are open for surgical management of this condition like Mitral valve replacement (MVR), Closed Mitral Commisurotmy and Open Mitral Valvotomy. Mitral Valve Replacement may be done with either mechanical prosthetic valve or Bioprosthetic Valve Autologous transfer of Pulmonary valve (ROSS II PROCEDURE) has also been done for this condition.

Cesjnvar[8] et al has reported higher early mortality among his series of 382 patients who underwent MVR for Mitral Valvular disease with Pulmonary Hypertension. But the late mortality was no different among patients with or without Pulmonary Hypertension. Aris[9] et al has confirmed this finding with his series of 88 patients. Perioperative mortality with patients having Suprasystemic Pulmonary Arterial pressures were 5 times more than with normal or sub systemic pressures.

In view of this fact subjecting patients with Mitral Valvular heart disease for earlier surgical intervention would ameliorate the condition before developing severe Pulmonary Hypertension.

PERSISTENT PHT FOLLOWING MVR

Following MVR majority of the patients have regression of PAP, yet Pulmonary Vascular hypertension remains unchanged in significant proportion of patients[19]. Patients with Suprasystemic PAP continue to have persistently elevated PVR. Patients with irreversible PHT after MVR are found to have early mortality. Compared to patients who have AF, regaining sinus rhythm after MVR results in reduction in PAP.

Patient –prosthetic mismatch contributes significantly to the persistence of Pulmonary Hypertension after MVR. Recent studies had demonstrated the correlation between

persistent PHT and patient –prosthesis mismatch and associated poor prognosis. Patient –prosthesis mismatch occurs with both mechanical and Bioprosthesis Valves.

TRICUSPID REGURGITATION AND PHT

Functional TR occurs following severe PHT in a Rheumatic Mitral Valvular heart disease. There are conflicting reports regarding the resolution of TR following Mitral Valve Replacement. Persistence of TR may contribute to the mortality and morbidity following surgeries done for mitral valve disease. Some times simultaneous TR repair is recommended during surgeries done for mitral valve disease. Patients with severe PHT show regression of TR following MVR and other surgeries for mitral valve disease.

PERIOPERATIVE MANAGEMENT OF PHT PATIENTS

Perioperative mortality is higher with patients having severe PHT and mitral valvular heart disease. But late survival curves are similar for patients with or without Pulmonary Hypertension, therefore effective perioperative management of severe Pulmonary Hypertension would help in the late survival of these patients and there would be no significant difference in the Kaplan-Meier survival curves.

Various drugs are available for managing these patients perioperatively like milrinone, nesiridite, inhaled nitric oxide and prostacyclin and on the horizon we also have sildanefil and endothelin antagonist like bosentan etc which has been used both preoperatively and postoperatively .

Role of phenoxybenzamine has been found be useful in cases of Pulmonary Hypertension secondary to congenital heart disease and in the peri and postoperative periods of congenital heart disease correction especially with elevated Pulmonary vascular resistance .

TREATMENT TARGETED AT PULMONARY VASCULATURE

So far traditionally treatment for mitral valvular heart disease focused only on cardiac physiology. Now the recent concepts are focusing more in the Pulmonary Vascular physiology following mitral valve disease and are due to the renewed interest in Cardio Pulmonary Hemodynamics. Madden[15] et al has recently found the role of phosphodiesterase 5 inhibitor (sildenafil) in a study conducted by him and found to have a positive role in treating these patients. The therapy has been found to be significantly tolerated and results are good as early as 8 weeks .Even exercise tolerance seems to be improved with this medicine.

PULMONARY VASCULAR RESISTANCE

Pulmonary vascular resistance has two components an organic element and dynamic element. Dynamic component is relieved immediately following reduction of LA pressures. Organic element due to changes in Pulmonary vascular changes regresses immediately or may take a long time to do. Kaul and colleagues reported on 30 patients with severe Pulmonary Hypertension which showed striking regression from a mean of 74mm Hg. Restudy an average of 5.5 years after MVR showed an average systolic pressure of 48mmHg and mean of 31 mmHg. This drop was largely due to sudden reduction of LA pressure and reversal of severe spastic Pulmonary vasoconstriction that accompanies left Atrial Hypertension. These changes in Pulmonary vascular resistance are a progressive regression and are the same for both stenosis and regurgitation. In older patients and in patients with AF the regression occurs less frequently.

CALCULATION OF PULMONARY ARTERY PRESSURE

Using Doppler we can measure the Pulmonary artery pressure using the peak Transtricuspid flow velocity (VMAX).

PAP = $4V\ max^2$ + RIGHT ATRIAL PRESSURE.

RAP is usually equal to jugular venous pressure measured clinically or from the Inferior Vena Cava diameter measured in Expiration and percentage collapse of IVC in Inspiration .

MITRAL STENOSIS

The mitral valve is made up of the annulus, anterior and posterior leaflets, and chordae, which attach the leaflets to their respective papillary muscles. A normally functioning valve allows blood to flow unimpeded from the left atrium to the left ventricle during diastole and prevents regurgitation during systole. Normal mitral valve function is dependent not only on the integrity of the underlying valvular structure, but on that of the adjacent myocardium as well.

Definition and Causes

Mitral stenosis (MS) refers to narrowing of the mitral valve orifice, resulting in impedance of filling of the left ventricle in diastole. It is usually caused by rheumatic heart disease. Less common causes include severe calcification of the mitral annulus, infective endocarditis, systemic lupus erythematosus, rheumatoid arthritis, and carcinoid heart disease.

Pathophysiology and Natural History

Patients with MS typically present more than 20 years after an episode of rheumatic fever. Single or recurrent bouts of Rheumatic Carditis cause progressive thickening, scarring, and calcification of the mitral leaflets and chordae. Fusion of the commissures and chordae decreases the size of the mitral opening. This obstruction results in the development of a pressure gradient across the valve in diastole and causes an elevation in left atrial and Pulmonary venous pressures. Elevated left atrial pressures lead to left atrial enlargement, predisposing the patient to Atrial Fibrillation and Arterial Thromboembolism. Elevated Pulmonary Venous pressure results in

Pulmonary Congestion and Pulmonary Edema. In advanced mitral stenosis, patients develop Pulmonary Hypertension and right-sided heart failure.

Signs and Symptoms

Patients with mitral stenosis may present with Exertional Dyspnea, fatigue, atrial arrhythmias, embolic events, angina-like chest pain, Hemoptysis, or even right-sided heart failure. Previously asymptomatic or stable patients may decompensate acutely during exercise, emotional stress, pregnancy, infection, or with uncontrolled atrial fibrillation.

The characteristic findings of MS on auscultation are an accentuated first heart sound, an opening snap, and a mid-diastolic rumble. The first heart sound may be diminished in intensity if the valve is heavily calcified, with limited mobility. If the patient is in sinus rhythm, there is presystolic accentuation of the murmur during atrial contraction. With increasingly severe stenosis, the duration of the murmur increases and the opening snap occurs earlier during diastole as a result of higher left atrial pressure. There is accentuation of P_2 when Pulmonary Hypertension is present. If flow across the mitral valve is reduced because of heart failure, Pulmonary Hypertension, or aortic stenosis the murmur of mitral stenosis may be reduced in intensity or may be inaudible.

Left Atrial Myxoma may be distinguished from MS by the presence of a “tumor plop” versus an opening snap in early diastole.

Diagnosis

On chest radiography, the characteristic findings of mitral stenosis are pulmonary congestion, enlargement of the main Pulmonary arteries, and enlargement of the left atrium without cardiomegaly. An electrocardiogram (ECG) may reveal evidence of left atrial enlargement, Atrial Fibrillation or, in advanced disease, right ventricular hypertrophy consistent with Pulmonary Hypertension.

Two-dimensional (2D) and Doppler echocardiography is indicated for all patients with suspected MS to confirm the diagnosis and determine its severity (Class I indication). Characteristic findings of MS include valve thickening, restricted valve opening, anterior leaflet doming, and fusion of the leaflets at the commissures. The mean pressure gradient across the mitral valve on Doppler echocardiography (echo) in MS is at least 5 mm Hg; in severe stenosis, it is usually higher than 10 mm Hg. Because the gradient across the mitral valve is flow dependent, the severity of MS is more accurately defined by the mitral valve area (MVA). The normal valve area is 4 to 5 cm^2. In mild mitral stenosis, the MVA is 1.5 to 2 cm^2, in moderate stenosis it is 1 to 1.5 cm^2, and in severe stenosis it is less than 1 cm^2. The valve area may be measured by tracing the mitral valve opening in cross section by 2D echo. Alternatively, the MVA is calculated using the pressure half-time (P ×-½t), which is the amount of time it takes for the transmitral pressure to fall to one half its initial value (MVA = 220/[P ×- ½t]).

Echocardiography also allows assessment of Pulmonary artery pressures, detection of other valve disease, visualization of left atrial thrombus, and identification of important differential diagnoses, such as Left Atrial Myxoma. Tran esophageal echo is superior to transthoracic echo at identifying left atrial thrombus in patients who are being considered for Percutaneous Mitral Balloon Valvotomy or Cardio Version. Stress echocardiography may be helpful if there is a discrepancy between a patient's severity of symptoms and the baseline hemodynamic data. An exercise mean transmitral gradient of more than 15 mm Hg and peak right ventricular systolic pressure of more than 60 mm Hg indicate hemodynamically significant MS.

Cardiac catheterization is not necessary in all cases but, like stress echocardiography, may be helpful in characterizing the severity of mitral stenosis when there is a discrepancy between symptoms and findings on echocardiography.

Treatment

Medical Treatment

Medical therapy has no role in altering the natural history or delaying the need for surgery in patients with MS. Medical treatment is directed toward alleviating Pulmonary Congestion with diuretics, treating Atrial Fibrillation, and anticoagulating patients who are at increased risk of arterial embolic events.

Development of Atrial Fibrillation frequently leads to an acute deterioration in patients with mitral stenosis. The rapid ventricular response results in a decrease in the diastolic filling time. Beta blockers, calcium channel blockers, or digoxin may be used to control ventricular rate. An attempt to restore sinus rhythm with direct current electrical cardioversion or antiarrhythmic drugs may be considered. Anticoagulation with warfarin is indicated to prevent thromboembolism when Atrial Fibrillation is present, if there is a prior history of thromboembolism, or a thrombus is detected in the left atrium (Class I). Although controversial, anticoagulation may also be considered if the left atrium is markedly dilated (5.0 to 5.5 mm) or if there is spontaneous contrast on echocardiography (Class II b).

Antibiotic therapy is important for the secondary prevention of Rheumatic Carditis. Patients with a history of rheumatic fever are at high risk of recurrence. Long-term secondary prophylaxis, preferentially with penicillin, is therefore recommended for all patients with a history of rheumatic fever or suspected rheumatic valve disease. The duration of prophylaxis depends on a number of factors, including the time lapsed since the last attack, the age of the patient, the presence or absence of cardiac involvement, and the patient's risk of exposure to streptococcal infections. Routine antibiotic prophylaxis for endocarditis is no longer recommended for patients with mitral stenosis.

Surgery

Three invasive options are available for patients with MS: (1) Percutaneous Mitral Balloon Valvotomy (PMBV); (2) Surgical Mitral Commissurotomy; and (3) Mitral Valve Replacement (MVR). In experienced centers, PMBV is the initial procedure of choice and should be considered for (1) symptomatic patients (NYHA functional Classes II to IV) with moderate or severe MS (Class I) and (2) asymptomatic patients with moderate or severe MS and Pulmonary Hypertension (Class I). PMBV is a catheter-based technique in which a balloon is inflated across the stenotic valve to split the fused commissures and increase the valve area. The MVA typically doubles in size, and hemodynamic as well as clinical improvements are seen immediately. The results are comparable with those achieved with open Mitral Commissurotomy, but it is less invasive and less costly. The Mitral Valve Morphology is an important predictor of successful balloon valvotomy. Severe valve calcification or significant involvement of the subvalvular apparatus on echocardiography before PMBV is associated with a higher complication rate and a greater risk of recurrence. In addition, balloon valvotomy should not be performed in patients who have left atrial thrombus or more than 2+ (moderate) mitral regurgitation, because the degree of mitral regurgitation usually increases following the procedure. Complications of Balloon Mitral Valvotomy include severe Mitral Regurgitation (3%), Thromboembolism (3%), and residual Atrial Septal Defect with significant shunting (less than 5%). Mortality with the procedure is lower than 1% in experienced hands. At 7 years after balloon valvotomy, 50% to 69% of patients remain free of cardiovascular events and up to 90% of patients remain free of reintervention. However, both Balloon Valvotomy and Surgical Commissurotomy are palliative procedures and, in most cases, further intervention is eventually required, usually in the form of a Mitral Valve Replacement.

In patients with calcified valves that cannot be treated by valvotomy or commissurotomy, or in those with significant mitral regurgitation that is not suitable for repair, mitral valve replacement may be necessary. The threshold for Mitral Valve Surgery (commisurotmy or MVR) is higher than for PMBV in patients with Mitral Stenosis, and commisurotmy or repair is preferable to MVR, if feasible. Surgery for

moderate to severe Mitral Stenosis is indicated for symptomatic patients (New York heart association [NYHA] functional class iii or iv) where PMBV is unavailable or contraindicated (class i). MVR may also be considered for patients with severe MS and severe Pulmonary Hypertension with NYHA functional classes i or ii symptoms who are not candidates for PMBV or Mitral Valve Repair (class iia). Both mechanical and biologic prostheses are used for MVR; the choice of valve often depends on factors such as age, need for Concomitant Anticoagulation, and Left Ventricular (LV) size. Morbidity and mortality are higher with prosthetic valve replacement than with surgical or balloon valvotomy**.**

Definition and Causes

Mitral Regurgitation (MR) is leakage of blood from the left ventricle into the left atrium during systole. It is caused by various mechanisms related to structural or functional abnormalities of the mitral apparatus, adjacent myocardium, or both. The most common causes of mitral regurgitation are rheumatic heart disease, myxomatous degeneration, chordal rupture, infective endocarditis, coronary artery disease, and cardiomyopathy .

Pathophysiology and Natural History

Significant MR leads to volume overload of the left ventricle, because it has to accommodate both the stroke volume and regurgitant volume with each heartbeat. To compensate, the left ventricle dilates and becomes hyperdynamic. In acute severe MR, the left atrial and pulmonary venous pressures increase quickly, leading to pulmonary congestion and Pulmonary Edema. In chronic MR, a gradual increase in left atrial size and compliance compensate so that left atrial and pulmonary venous pressures do not increase until late in the course of the disease. Progressive left ventricular dilation eventually leads to an increase in afterload, contractile dysfunction, and heart failure. Left atrial enlargement predisposes the patient to Atrial Fibrillation and Arterial

Thromboembolism. In long-standing MR, patients may develop Pulmonary Hypertension and right-sided heart failure.

Signs and Symptoms

Patients with chronic, severe mitral regurgitation may remain asymptomatic for years because the regurgitant volume load is well tolerated as a result of compensatory ventricular and atrial dilation. When symptoms do develop, the most common are dyspnea, fatigue, orthopnea, paroxysmal nocturnal dyspnea, and palpitations caused by Atrial Fibrillation. Acute severe MR, as occurs with chordal rupture or papillary muscle rupture, is almost always symptomatic because the sudden regurgitant volume load in the nondilated left ventricle and atrium leads to Pulmonary venous hypertension and congestion.

The characteristic finding in a patient with MR is a blowing holosystolic murmur heard best at the cardiac apex. When ventricular enlargement is present, the apical impulse may be diffuse and laterally displaced, and a third heart sound may be heard.

Diagnosis

The chest radiograph demonstrates left atrial enlargement and cardiomegaly. Two-dimensional and Doppler echocardiography is indicated for all patients with suspected mitral regurgitation to confirm its presence and determine its severity (Class I). Two-dimensional echocardiography usually reveals the cause (e.g., the presence of myxomatous mitral valve disease and leaflet prolapse or evidence of underlying dilated cardiomyopathy). Evaluation of the severity of Mitral Regurgitation on echocardiography requires an integrated assessment of several parameters, including regurgitant jet size by Color Doppler, Regurgitant Jet Density by continuous-wave (CW) Doppler, and Pulmonary vein and Mitral Valve inflow by pulse-wave (PW) Doppler. Newer applications of Doppler echocardiography allow quantitative measurement of mitral regurgitation, including the regurgitant volume and the

regurgitant orifice area (ROA)—that is, the area through which the valve leaks in systole. In asymptomatic patients with significant mitral regurgitation, serial echocardiography every 6 to 12 months to assess LV size and systolic function is important for optimal timing of surgery (Class I). Transesophageal echocardiography is indicated for patients who are not adequately imaged by transthoracic echocardiography and before surgery to assess feasibility for repair (Class I). Stress echocardiography may be useful to assess exercise tolerance and the response of mitral regurgitation severity, Pulmonary pressure, and contractile reserve to exercise in asymptomatic patients with significant MR (Class IIa).

Cardiac catheterization is no longer routinely performed to evaluate mitral regurgitation severity, but it is indicated for those patients in whom noninvasive test results are inconclusive, and also to detect concomitant Coronary Artery Disease (CAD) in patients undergoing mitral valve surgery (Class I).

Treatment

Medical Treatment

In patients with acute severe MR, afterload reduction with intravenous nitroprusside and nitroglycerin reduces the regurgitant fraction and Pulmonary pressures. Placement of an intra-aortic balloon pump also helps stabilize these patients. However, these are temporary measures before urgent mitral valve repair or replacement. In patients with chronic asymptomatic Mitral Regurgitation caused by primary valve disease, there is no evidence for the routine use of medication in delaying the need for surgery or preventing left ventricular dysfunction. The management of these patients is focused on deciding on the appropriate timing of surgery, before the development of irreversible left Ventricular Dysfunction. Patients should be followed up every 6 to 12 months to assess for symptoms and to measure left ventricular size, function, and severity of MR by echocardiography (Class I).

In patients with ischemic heart disease or dilated cardiomyopathy, mitral regurgitation indicates a poor prognosis. MR in these patients is called functional mitral regurgitation and is caused by global or regional changes in left ventricular geometry as well as annular dilation. Functional MR is primarily treated medically with antihypertensive therapy, Angiotensin Converting Enzyme (ACE) inhibitors, beta blockers, diuretics, and antianginal therapies when mitral regurgitation is worsened by acute ischemia. Biventricular Pacing has also been shown to decrease the degree of mitral regurgitation in dilated cardiomyopathy.

Routine antibiotic prophylaxis for endocarditis is no longer recommended for patients with mitral regurgitation.

Surgery

Surgery is indicated for (1) symptomatic patients with severe primary MR (Class I) and (2) asymptomatic patients with severe primary MR and evidence of LV dysfunction (Class I). Optimal timing of mitral valve surgery is challenging in asymptomatic patients because the actual contractile function of the left ventricle is difficult to measure. The standard indications for surgery in asymptomatic patients are an LV end-systolic dimension of more than 4.0 cm and a resting LV ejection fraction of less than 60% (Class I). Other indications in asymptomatic patients include Pulmonary Hypertension or development of Atrial Fibrillation (Class IIa). In addition, mitral valve repair may be undertaken in experienced surgical centers for asymptomatic patients with severe MR, but without evidence of LV dilation or dysfunction, for which the likelihood of a successful repair is greater than 90% (Class IIa). Most asymptomatic patients with severe MR develop symptoms, LV dysfunction, or both over long-term follow-up. One retrospective study showed an increased risk of cardiac death (4%/year) in patients with severe mitral regurgitation based on an ROA of more than 0.4 cm^2. However, another recent prospective study has shown that careful follow-up of patients with severe MR and timing of surgery based on symptoms, LV dysfunction, development of Atrial Fibrillation, or Pulmonary Hypertension is associated with an excellent patient outcome.

In patients with severe functional mitral regurgitation, surgery may be considered for severe symptoms despite medical therapy. Patients with ischemic MR may improve with coronary bypass surgery if significant ischemia or myocardial viability is present. In many coronary bypass patients with MR, concomitant mitral valve repair with an undersized annuloplasty ring is performed. Patients with severe left ventricular dysfunction and significant MR were once believed to be poor surgical candidates, but recent studies have shown an acceptable operative risk. Symptoms usually improve, although a survival benefit has not been demonstrated.

The two available surgical options are mitral valve repair and mitral valve replacement. Mitral valve repair is the procedure of choice in the surgical management of MR caused by degenerative valve disease and in some cases of MR caused by infective endocarditis and ischemic heart disease. Repair offers several advantages over replacement, including lower operative time and long-term mortality, better preservation of LV function, a lower risk of subsequent infective endocarditis, and no need for long-term anticoagulation. Reoperation rates for mitral valve repair and replacement are similar, occurring at a rate of 1% to 2% per year. On the other hand, repair is technically more difficult than replacement, and many cases of mitral regurgitation are not amenable to valve repair. Percutaneous mitral valve repair is currently being investigated. The techniques involved include a clip that joins the mitral leaflets at their midpoint and an annuloplasty ring delivered via the coronary sinus.

Aims & Objectives

AIMS AND OBJECTIVES

- A Prospective study to analyze preoperative and postoperative Pulmonary Hypertension following mitral valvular heart disease using preoperative echocardiogram, intraoperative pressure studies and postoperative echocardiogram.

- To analyze the reduction in Pulmonary Hypertension following mitral valve replacement during patient follow up post surgically.

- To study the effect of pharmacological agents in reduction of Pulmonary Hypertension preoperatively and postoperatively in mitral valvular heart disease.

Materials & Methods

MATERIALS AND METHODS

A total of 265 patients were taken in this prospective study who came to the hospital with NYHA Class II-IV symptoms and with Mitral valvular heart lesions comprising of Mitral stenosis and Mitral Regurgitation with Pulmonary Hypertension from period of July 2007 –July 2009.

A detailed clinical examination and investigations were done on these patients and were categorized according to the study. All patients were prepared and consented before surgery as per the protocol and were operated under same conditions. All of them underwent Mitral valve replacement using St Jude's medical valve using cardiopulmonary bypass support.

A detailed clinical findings and preoperative values of these 265 patients are recorded over a structured proforma (Annexure) for all the patients.

All the patients are classified according to their age, sex, diagnosis, preoperative, intraoperative and post operative echo findings and result after surgery. The results thus obtained are tabulated and statistically analyzed and conclusions drawn thereafter.

Observations & Results

OBSERVATIONS AND RESULTS

265 Patients with mitral valvular heart disease and Pulmonary Hypertension who were admitted in government general hospital during July 2007-july 2010 were classified into different groups and tabulated according to different variables as follows:

AGE GROUP Vs PULMONARY HT SEVERITY PRE OPERATIVE

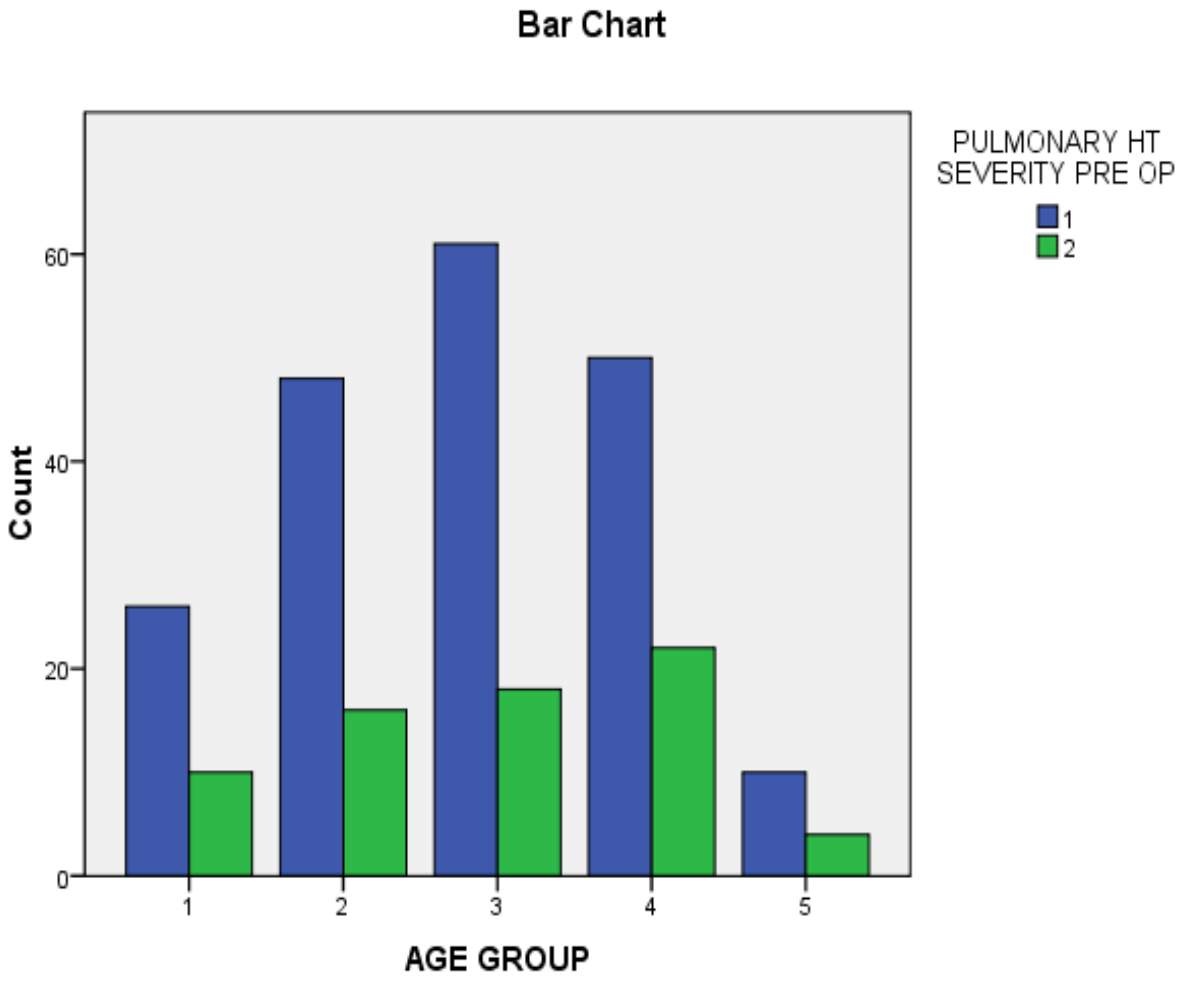
			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
AGE GROUP	1 (10-19)	Count	26	10	36
		% within PULMONARY HT SEVERITY PRE OP	13.3%	14.3%	13.6%
		% of Total	9.8%	3.8%	13.6%
	2 (20-29)	Count	48	16	64
		% within PULMONARY HT SEVERITY PRE OP	24.6%	22.9%	24.2%
		% of Total	18.1%	6.0%	24.2%
	3 (30-39)	Count	61	18	79
		% within PULMONARY HT SEVERITY PRE OP	31.3%	25.7%	29.8%
		% of Total	23.0%	6.8%	29.8%

Cont..

			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
	4 (40-49)	Count	50	22	72
		% within PULMONARY HT SEVERITY PRE OP	25.6%	31.4%	27.2%
		% of Total	18.9%	8.3%	27.2%
	5 (>50)	Count	10	4	14
		% within PULMONARY HT SEVERITY PRE OP	5.1%	5.7%	5.3%
		% of Total	3.8%	1.5%	5.3%
	Total	Count	195	70	265
		% within PULMONARY HT SEVERITY PRE OP	100.0%	100.0%	100.0%

Out of 265 patients analyzed 195 (73.58%) had severe Pulmonary Hypertension and remaining 70 patients (26.42 %) had moderate to mild Hypertension. Out of 265 patients, age group which had affected maximum with PHT was found to be in the 3rd to 4th decade which is about 31.3% which is a significant productive group. Next group to be affected by this severe PHT was found to be in the 4th to 5th decade (18.9 %).Age group which had affected the least was found in the 5th decade and beyond.

Chi-Square Tests			
	Value	df	Asymmetrical. Sig. (2-sided)
Pearson Chi-Square	1.304[a]	4	.861
On performing the chi-square test there was no significant correlation between the age group and severe Pulmonary Hypertension.			



1) 10-19 Yrs
2) 20-29 Yrs
3) 30-39 Yrs
4) 40-49 Yrs
5) >50 Yrs

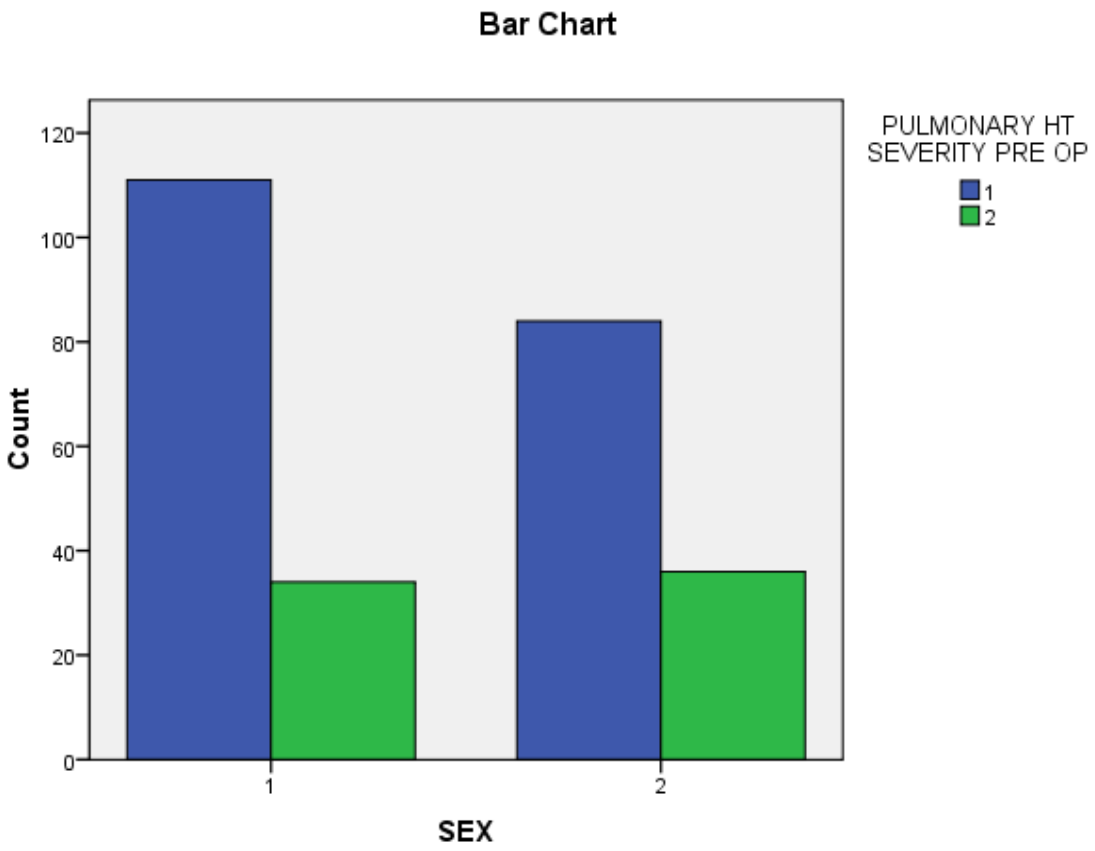
SEX DISTRIBUTION

SEX Vs PULMONARY HT SEVERITY PRE OPERATIVE					
			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
SEX	1 (Female)	Count	111	34	145
		% within PULMONARY HT SEVERITY PRE OP	56.9%	48.6%	54.7%
		% of Total	41.9%	12.8%	54.7%
	2 (Male)	Count	84	36	120
		% within PULMONARY HT SEVERITY PRE OP	43.1%	51.4%	45.3%
		% of Total	31.7%	13.6%	45.3%
Total		Count	195	70	265
		% within PULMONARY HT SEVERITY PRE OP	100.0%	100.0%	100.0%
		% of Total	73.6%	26.4%	100.0%

According to this study the female patients were affected more with severe Pulmonary Hypertension which constitutes about (41.9%) and male patients constituted about 31.7% out of the 195 patients who had severe Pulmonary Hypertension. On an average about 73.6% of patients had severe Pulmonary Hypertension taking both males and females into consideration. The distribution for mild to moderate disease was found to be equal.

Statistical Method	Value	df	Asymmetric Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.450[a]	1	.229		

On performing the chi-square test there was no significant statistical correlation between sex and Pulmonary Hypertension.



1) MALE 2) FEMALE

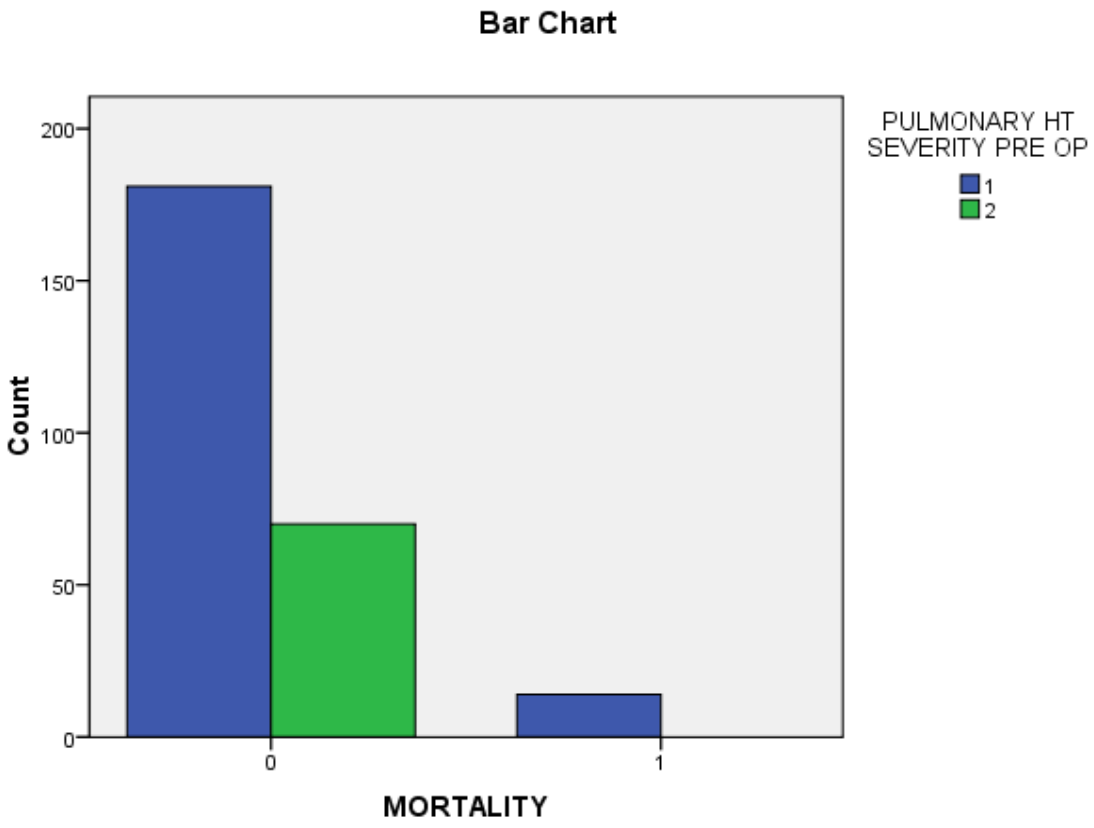
MORTALITY AND PULMONARY HYPERTENSION

PULMONARY HT SEVERITY PRE OPERATIVE			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
MORTALITY	0	Count	181	70	251
		% within PULMONARY HT SEVERITY PRE OP	92.8%	100.0%	94.7%
		% of Total	68.3%	26.4%	94.7%
	1	Count	14	0	14
		% within PULMONARY HT SEVERITY PRE OP	7.2%	.0%	5.3%
		% of Total	5.3%	.0%	5.3%
	Total	Count	195	70	265
		% within PULMONARY HT SEVERITY PRE OP	100.0%	100.0%	100.0%
		% of Total	73.6%	26.4%	100.0%

On analyzing mortality in relation to Pulmonary Hypertension there were 14 deaths (5.3%) out of total number of 265 patients and which includes about 7.2% of 195 patients known to have severe Pulmonary Hypertension. About 94.7% patients had good postoperative outcome following this surgery done for severe PHT associated with Rheumatic Mitral valve disease.

Statistical Method	Value	df	Asymmetric. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	5.306[a]	1	.021		

On performing the chi-square test there was a significant correlation between severe Pulmonary Hypertension and mortality which is a well known factor and this study also confirms the hypothesis.



1- No Mortality

2-Mortality

PATIENTS IN NYHA POST MVR (NYHA Vs PULMONARY HT SEVERITY PRE OPERATIVE)

PULMONARY HT SEVERITY PRE OPERATIVE			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
NYHA	1	Count	6	48	54
		% within PULMONARY HT SEVERITY PRE OP	3.3%	68.6%	21.5%
		% of Total	2.4%	19.1%	21.5%
	2	Count	172	22	194
		% within PULMONARY HT SEVERITY PRE OP	95.0%	31.4%	77.3%
		% of Total	68.5%	8.8%	77.3%
	3	Count	3	0	3
		% within PULMONARY HT SEVERITY PRE OP	1.7%	.0%	1.2%
		% of Total	1.2%	.0%	1.2%
	Total	Count	181	70	251
		% within PULMONARY HT SEVERITY PRE OP	100.0%	100.0%	100.0%

PULMONARY HT SEVERITY PRE OPERATIVE			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
NYHA	1	Count	6	48	54
		% within PULMONARY HT SEVERITY PRE OP	3.3%	68.6%	21.5%
		% of Total	2.4%	19.1%	21.5%
	2	Count	172	22	194
		% within PULMONARY HT SEVERITY PRE OP	95.0%	31.4%	77.3%
		% of Total	68.5%	8.8%	77.3%
	3	Count	3	0	3
		% within PULMONARY HT SEVERITY PRE OP	1.7%	.0%	1.2%
		% of Total	1.2%	.0%	1.2%
	Total	Count	181	70	251
		% within PULMONARY HT SEVERITY PRE OP	100.0%	100.0%	100.0%
		% of Total	72.1%	27.9%	100.0%

About 77.3% of patients were in NYHA classification II following this surgery and about 21.5% were in NYHA class I followed by 1.2% in NYHA Class III.

REDUCTION OF PULMONARY HYPERTENSION

T-Test

Group Statistics

	SEX	N	Mean	Std. Deviation	Std. Error Mean
PRE-OPERATIVE PULMONARY HT	1	145	72.15	19.993	1.660
	2	120	69.34	20.756	1.895
0-MONTHS PULMONRY HT	1	145	45.50	11.775	.978
	2	120	46.77	10.744	.981
3-MONTHS PULMONARY HT	1	134	37.56	6.245	.539
	2	117	39.58	8.237	.762
6-MONTHS PULMONARY HT	1	134	29.78	5.356	.463
	2	117	30.97	6.515	.602

On analyzing the data the Pulmonary Hypertension was reduced to a mean of approximately 46.14mmHG immediately following surgery and significant reduction of about 30.38 mmHG after 6 months of surgery. At 3 months follow up the average reduction in PHT was about 38.57mmHG .This analysis showed that the reduction in PHT had a gradual course after an initial reduction from a mean PHT value of 70.75 mmHG preoperative values. This gives us a clue that the reduction in Pulmonary Hypertension is a gradual one and needed change in organic level as well as in the dynamic level which takes some time to achieve.

Independent Samples Test

		t-test for Equality of Means		
		df	Sig.(2-tailed)	Mean Difference
PRE OP PULMONARY HT	Equal variances assumed	263	.264	2.810
	Equal variances not assumed	250.067	.266	2.810
0-MONTHS PULMONRY HT	Equal variances assumed	263	.364	-1.270
	Equal variances not assumed	260.472	.360	-1.270
3-MONTHS PULMONARY HT	Equal variances assumed	249	.028	-2.021
	Equal variances not assumed	214.527	.031	-2.021
6-MONTHS PULMONARY HT	Equal variances assumed	249	.111	-1.198
	Equal variances not assumed	224.980	.116	-1.198

On performing the T-test on the given statistics there was a statistical significance in patients who had a reduction in Pulmonary Hypertension after 3 months postoperatively from the preoperative value.

Preoperative Hemodynamic in Patients with Pulmonary Hypertension

Variable	Severe PHT
Systolic PAP (mm Hg) [range]	84.5 [75–105]
Mean PAP (mm Hg) [range]	70.88 [51–90]

An average systolic pressure of 84.5mmHg and a mean PAP of 70.88 mmHg were found in this study.

INTRAOPERATIVE PHT

S.NO	MALE	FEMALE
PAP (MEAN)	62.8mmHg	73.6 mmHg

An average mean PAP of 62.8mmHg and 73.6mmHg were observed in male and female patients respectively with severe Pulmonary Hypertension intraoperatively.

POST MVR PHT (6 MONTHS)

S.NO	MALE	FEMALE
MEAN PAP	30.97mmHg	29.78mmHg

An average 30.97 mmg and 29.78mmHg were recorded as mean PAP in male and female patients respectively following Mitral valve replacement after an observation of 6 months.

PAP AT DAY 0

S.NO	MALE	FEMALE
MEAN PAP	46.77mmHg	45.5mmHg

At day zero the average reduction in PAP were found to be about 46.77mmHg and 45.5mmHg in both male and female patients respectively following MVR. This is found to be significant observation as the immediate drastic reduction in PHT as we expect following valve replacement does not occur as anticipated and gives us obvious clues as to the reasons behind the reduction in Pulmonary arterial pressures.

IMMEDIATE POSTOPERATIVE DEATH

S.NO	MALE	FEMALE
DEATH	04	10

An average of 7.07 %% of death in this study showed death in the immediate postoperative period which includes day 0 and day 1in patients with severe Pulmonary Hypertension .

MEAN ACC TIME AND CPB TIME

S.NO	ACC	CPB
LEFT ATRIAL APPROACH	56.6MINS	84 .56 MINS
SEPTAL APPROACH	68.2 MINS	100.68MINS

This table shows the average Aortic Cross Clamp time and Cardiopulmonary Bypass Time needed for the procedure. It is evident from the table that the septal approach takes a longer ACC and CPB time on comparison.

In all the 265 patients who underwent MVR, 195 of them had severe Pulmonary Hypertension. All of them had the classical left atrial approach through Sondergaard's groove incision except 11 cases which was approached through the septum after opening the right atrium.

All cases were opened through the standard median sternotomy and were done using cardiopulmonary bypass utilizing cardioplegic arrest to open the chamber (s).

In about 195 out of 265 patients (73.58%), MVR was done with either partial are complete chordal preservation. All the patients had St Jude's valve placed as the mechanical prosthetic valve. Except for the 14 deaths which occurred due to low cardiac output failures all other cases were weaned of the ventilator by day 1 and from the inotropic supports by day 2 or day 3.

Those patients who had severe Pulmonary Hypertension who underwent MVR were followed up using echo at day 0 ,3 months and 6 months later and results thus obtained showed an early reduction in about 21.16% of males and 27.35% of females .About 79.84% of male patients and 72.65% of female patients showed a trend of reduction in Pulmonary Hypertension in about 3- 6 months time as compared to those patients who showed an immediate reduction in Pulmonary Hypertension following mitral valve replacement for mitral valvular heart disease with severe Pulmonary Hypertension .

Discussions

DISCUSSION

Pulmonary Hypertension (PH or PHT) is an increase in blood pressure in the Pulmonary artery, Pulmonary vein, or Pulmonary capillaries, together known as the lung vasculature, leading to shortness of breath, dizziness, fainting, and other symptoms, all of which are exacerbated by exertion. Pulmonary Hypertension can be a severe disease with a markedly decreased exercise tolerance and heart failure. It was first identified by Dr. Ernst von Romberg in 1891. According to the most recent classification, it can be one of five different types: *arterial, venous, hypoxic, thromboembolic* or *miscellaneous*

SIGNS AND SYMPTOMS

Because symptoms may develop very gradually, patients may delay seeing a physician for years. Common symptoms are shortness of breath, fatigue, non-productive cough, angina pectoris, fainting or syncope, peripheral edema (swelling around the ankles and feet), and rarely hemoptysis (coughing up blood).

Pulmonary venous hypertension typically presents with shortness of breath while lying flat or sleeping (orthopnea or paroxysmal nocturnal dyspnea), while Pulmonary arterial hypertension (PAH) typically does not.

DIAGNOSIS

A physical examination is performed to look for typical signs of Pulmonary Hypertension, including a loud P2 (pulmonic valve closure sound), (Para) sternal heave, jugular venous distension, pedal edema, ascites, hepatojugular reflux, clubbing etc. Evidence of tricuspid insufficiency is also sought and, if present, is consistent with the presence of Pulmonary Hypertension.

Investigations include apart from the blood routine, an ECG, Echocardiogram to confirm diagnosis and quantify the severity, x-ray chest which shows the classical picture of dilated and prominent Pulmonary arteries with obliterated left cardiac shadow with straightening of left cardiac border. Arterial blood gas analysis or simple

bedside 02 saturation may give us the clue for the diagnosis. Biopsy of the lung is usually not indicated unless the Pulmonary Hypertension is thought to be due to an underlying interstitial lung disease. But lung biopsies are fraught with risks of bleeding due to the high intrapulmonary blood pressure. Blood BNP level is also being used now to follow progress of patients with Pulmonary Hypertension.

Cardiac catheterization is not routinely done nowadays to quantify Pulmonary Hypertension as newer Echocardiography studies throw us sufficient lights for the evidence and measurement of Pulmonary Hypertension.

Normal Pulmonary arterial pressure in a person living at sea level has a mean value of 12–16 mm Hg (1600–2100 Pa). Pulmonary Hypertension is present when mean Pulmonary artery pressure exceeds 25 mm Hg (3300 Pa) at rest or 30 mm Hg (4000 Pa) with exercise.

Mean Pulmonary artery pressure (mPAP) should not be confused with systolic Pulmonary artery pressure (sPAP), which is often reported on echocardiogram reports. A systolic pressure of 40 mm Hg typically implies a *mean* pressure more than 25 mm Hg. Roughly, mPAP = 0.61•sPAP + 2.

Vascular resistance is a term used to define the resistance to flow that must be overcome to push blood through the circulatory system. The resistance offered by the peripheral circulation is known as the systemic vascular resistance (SVR), while the resistance offered by the vasculature of the lungs is known as the Pulmonary vascular resistance (PVR).

Units for measuring vascular resistance are dynes·s·cm-5 or Pascal seconds per cubic meter (Pa.s/m^3). Pediatric cardiologists use hybrid reference units (HRU), also known as Wood units, as they were introduced by Dr. Paul Wood. To convert from dynes·s·cm-5 to Wood units you must divide by 80.

Normal Values For Vascular Resistance		
Systemic vascular resistance	1170 ± 270 dynes·s·cm^{-5}	117 ± 27 MPa.s/m^3
Systemic vascular resistance index	2130 ± 450 dynes·s·cm^{-5}·m^2	213 ± 45 MPa.s/m
Pulmonary vascular resistance	67 ± 30 dynes·s·cm^{-5}	6.7 ± 3 MPa.s/m^3

1 Calculation of resistance

The basic tenet of calculating resistance is that flow is equal to driving pressure divided by resistance.

CAUSES AND CLASSIFICATION

A 1973 meeting organized by the World Health Organization was the first to attempt classification of Pulmonary Hypertension. A distinction was made between primary and secondary PH, and primary PH was divided in the "arterial plexiform", "veno-occlusive" and "thromboembolic" forms. A second conference in 1998 at Évian-les-Bains also addressed the causes of secondary PH (i.e. those due to other medical conditions), and in 2003, the 3rd World Symposium on Pulmonary Arterial Hypertension was convened in Venice to modify the classification based on new understandings of disease mechanisms. The revised system developed by this group provides the current framework for understanding Pulmonary Hypertension. The system includes several improvements over the former 1998 Evian Classification system. Risk factor descriptions were updated, and the classification of congenital systemic-to Pulmonary shunts was revised. A new classification of genetic factors in PH was recommended, but not implemented because available data were judged to be inadequate.

The Venice 2003 Revised Classification system can be summarized as follows:

WHO Group I - Pulmonary arterial hypertension (PAH)

Idiopathic (IPAH)

Familial (FPAH)

Associated with other diseases (APAH): collagen vascular disease (e.g. scleroderma), congenital shunts between the systemic and Pulmonary circulation, portal hypertension, HIV infection, drugs, toxins, or other diseases or disorders

Associated with venous or capillary disease

WHO Group II - Pulmonary Hypertension associated with left heart disease

Atrial or ventricular disease

Valvular disease (e.g. mitral stenosis)

WHO Group III - Pulmonary Hypertension associated with lung diseases and/or hypoxemia

Chronic obstructive Pulmonary Disease (COPD), Interstitial Lung Disease (ILD)

Sleep-disordered breathing, alveolar hypoventilation

Chronic exposure to high altitude

Developmental lung abnormalities

WHO Group IV - Pulmonary Hypertension due to chronic thrombotic and/or embolic disease

Pulmonary Embolism in the proximal or distal Pulmonary Arteries

Embolization of other matter, such as tumor cells or parasites

WHO Group V - Miscellaneous

PATHOGENESIS

Whatever the initial cause**,** Pulmonary arterial hypertension (WHO Group I) involves the vasoconstriction or tightening of blood vessels connected to and within the lungs. This makes it harder for the heart to pump blood through the lungs, much as it is harder to make water flow through a narrow pipe as opposed to a wide one. Over time, the affected blood vessels become both stiffer and thicker, in a process known as

fibrosis. This further increases the blood pressure within the lungs and impairs their blood flow. In addition, the increased workload of the heart causes thickening and enlargement of the right ventricle, making the heart less able to pump blood through the lungs, causing right heart failure. As the blood flowing through the lungs decreases, the left side of the heart receives less blood. This blood may also carry less oxygen than normal. Therefore it becomes harder and harder for the left side of the heart to pump to supply sufficient oxygen to the rest of the body, especially during physical activity.

Pathogenesis in Pulmonary venous hypertension (WHO Group II) is completely different. There is no obstruction to blood flow in the lungs. Instead, the left heart fails to pumps blood efficiently, leading to pooling of blood in the lungs. This causes Pulmonary edema and pleural effusions.

In hypoxic Pulmonary Hypertension (WHO Group III), the low levels of oxygen are thought to cause vasoconstriction or tightening of Pulmonary arteries. This leads to a similar pathophysiology as Pulmonary arterial hypertension.

In chronic thromboembolic Pulmonary Hypertension (WHO Group IV), the blood vessels are blocked or narrowed with blood clots. Again, this leads to a similar pathophysiology as Pulmonary arterial hypertension.

TREATMENT

Treatment is determined by whether the PH is arterial, venous, hypoxic, thromboembolic, or miscellaneous. Since Pulmonary venous hypertension is synonymous with congestive heart failure, the treatment is to optimize left ventricular function by the use of diuretics, beta blockers, ACE inhibitors, etc., or to repair/replace the mitral valve or aortic valve.

In PAH, lifestyle changes, digoxin, diuretics, oral anticoagulants, and oxygen therapy are considered conventional therapy, but have never been proven to be beneficial in a randomized, prospective manner.

A number of agents has recently been introduced for primary and secondary PAH. The trials supporting the use of these agents have been relatively small, and the only measure consistently used to compare their effectivity is the "6 minute walking test". Many have no data on mortality benefit or time to progression.

VASOACTIVE SUBSTANCES

Many pathways are involved in the abnormal proliferation and contraction of the smooth muscle cells of the Pulmonary arteries in patients with Pulmonary arterial hypertension. Three of these pathways are important since they have been targeted with drugs — endothelin receptor antagonists, phosphodiesterase type 5 inhibitors[15], and prostacyclin derivatives.

Because inexpensive generic drugs for this disease are not widely available, the World Health Organization does not include them in its model list of essential medicines.

PROSTAGLANDINS

Prostacyclin (prostaglandin I_2) is commonly considered the most effective treatment for PAH. Epoprostenol (synthetic prostacyclin, marketed as Flolan) is given via continuous infusion that requires a semi-permanent central venous catheter. This delivery system can cause sepsis and thrombosis. Flolan is unstable, and therefore has to be kept on ice during administration. Since it has a half-life of 3 to 5 minutes, the infusion has to be continuous (24/7), and interruption can be fatal. Other prostanoids have therefore been developed. Treprostinil (Remodulin) can be given intravenously or subcutaneously, but the subcutaneous form can be very painful. An increased risk of sepsis with intravenous Remodulin has been reported by the CDC. Iloprost (Ilomedin) is also used in Europe intravenously and has a longer half life. Iloprost (marketed as Ventavis) is the only inhaled form of prostacyclin approved for use in the US and Europe. This form of administration has the advantage of selective deposition in the lungs with less systemic side effects. Oral and inhaled forms of

Remodulin are under development. Beraprost is an oral prostanoid available in Japan and South Korea. ENDOTHELIN RECEPTOR ANTAGONISTS

The dual (ET_A and ET_B) endothelin receptor antagonist bosentan (marketed as Tracleer) was approved in 2001. Sitaxentan, a selective endothelin receptor antagonist that blocks only the action of ET_A, has been approved for use in Canada, Australia, and the European Union, to be marketed under the name Thelin. Sitaxentan has not been approved for marketing by the U.S. Food and Drug Administration (FDA). A new trial to address the FDA's concerns had begun in 2008. A similar drug, ambrisentan is marketed as Letairis in U.S. In addition, another dual/nonselective endothelin antagonist, Actelion-1, from the makers of Tracleer, had entered clinical trials in 2008.

PHOSPHODIESTERASE TYPE 5 INHIBITORS

Sildenafil, a selective inhibitor of cGMP specific phosphodiesterase type 5 (PDE5), was approved for the treatment of PAH in 2005. It is marketed for PAH as Revatio. We had used this medication in about 73 (195) of the patients with severe Pulmonary Hypertension in our study.

ACTIVATORS OF SOLUBLE GUANYLATE CYCLASE

Soluble guanylate cyclase (sGC) is the intracellular receptor for NO. As of April 2009, the sGC activators cinaciguat and riociguat are undergoing clinical trials for the treatment of PAH

Treatment for hypoxic and miscellaneous varieties of Pulmonary Hypertension has not been established. However, studies of several agents are currently enrolling patients. Many physicians will treat these diseases with the same medications as for PAH, until better options become available. Such treatment is called off-label.

MONITORING

Patients are normally monitored through commonly available tests such as:

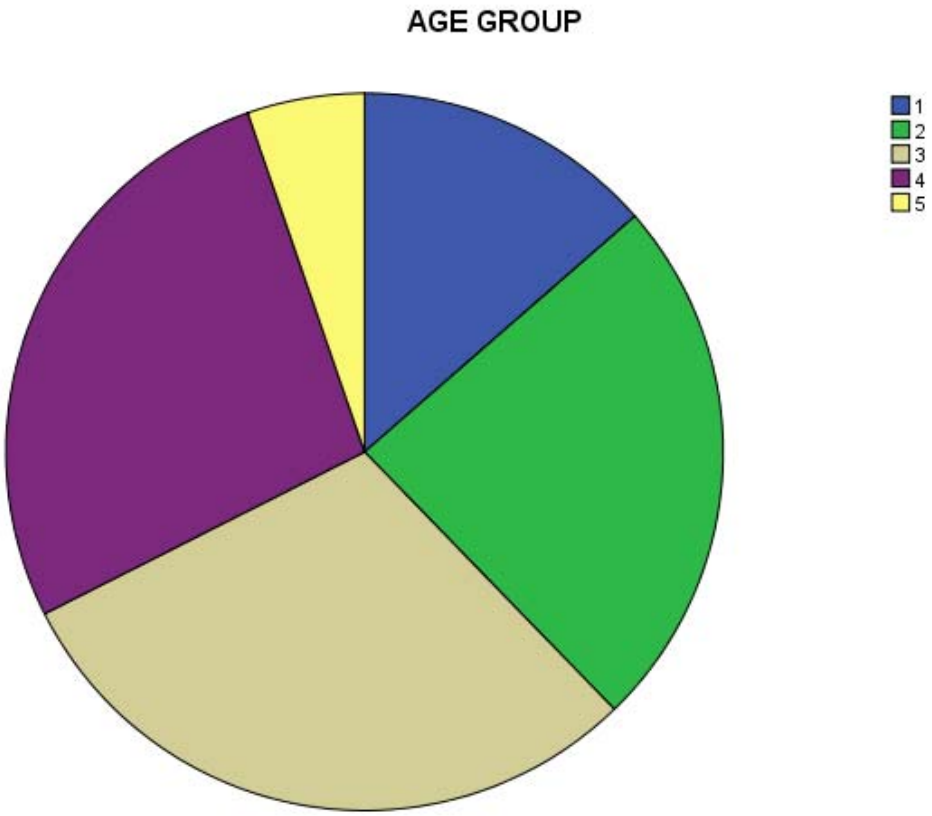
- Pulse oximetry,
- Arterial blood gas tests,
- Chest X-rays,
- Serial ECG tests,
- Serial echocardiography.

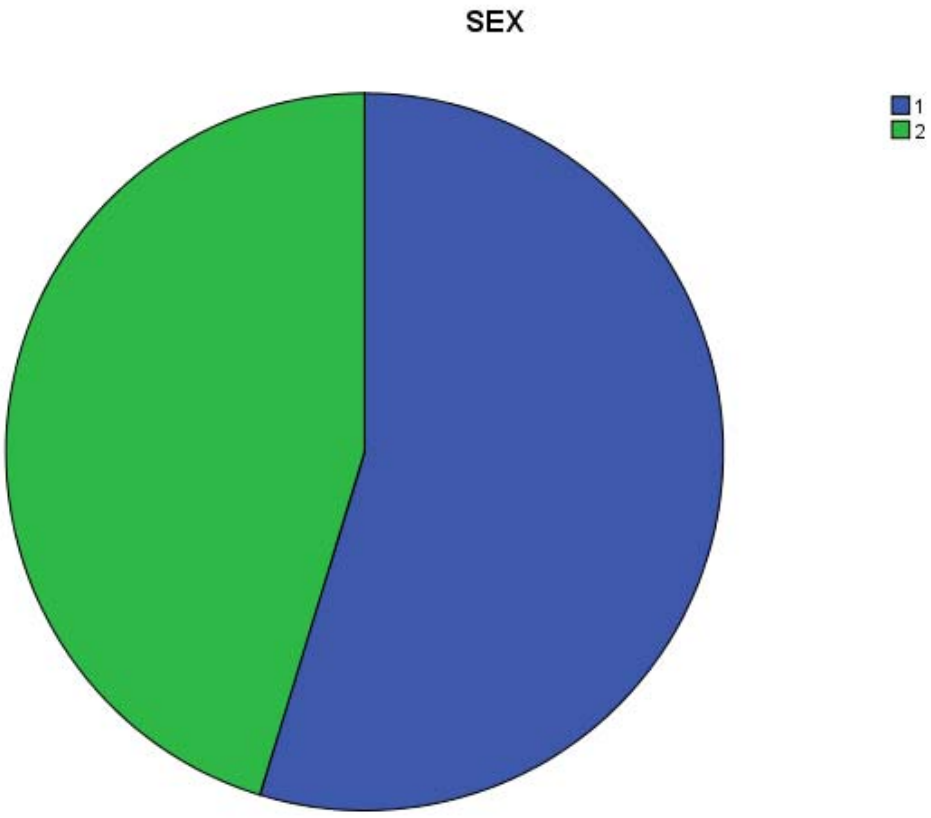
In all our 265 patients who underwent MVR 195 had severe Pulmonary Hypertension and females 111/195(56.9%) outnumbered the males in this diagnosis. The immediate reduction of Pulmonary Hypertension happened in only approximately 21 % of the patients and the remaining had persistent PHT which showed a fall progressively following a 6 months period suggesting the various factors which might be responsible for the Pulmonary Hypertension.

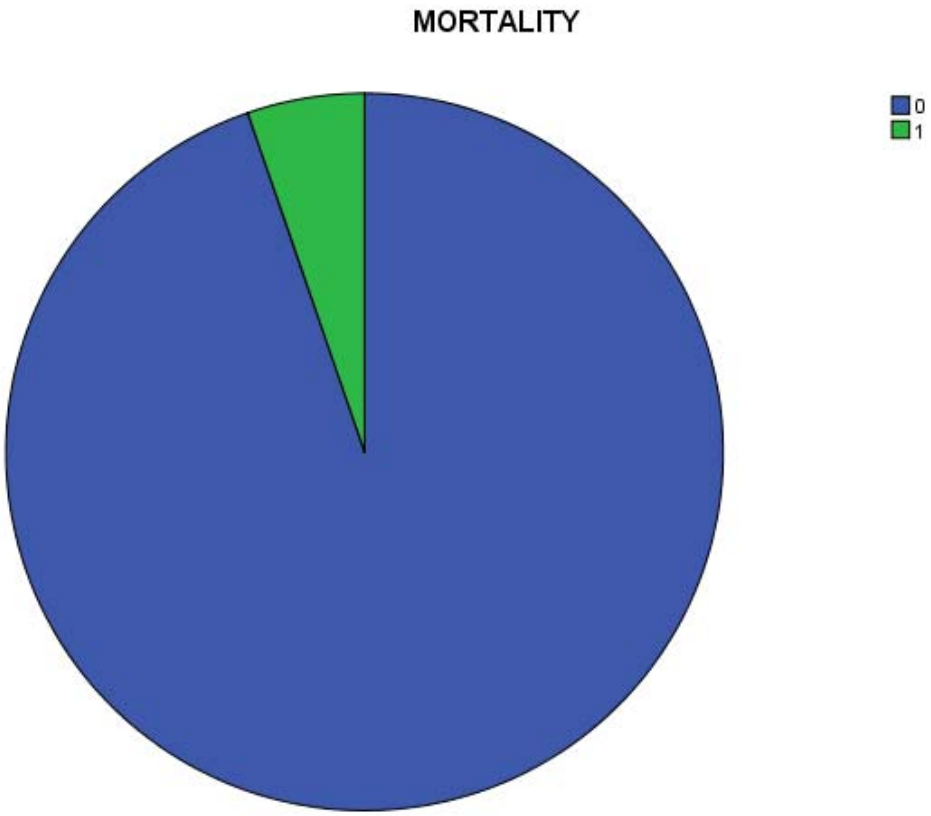
The average mortality noted in this study was comparatively less (7.2%) % than an expected value of about 10-15% by the previous studies. The difference might be because of the earlier decision to operate upon this severe group before irreversible factors sets in and also due to the better intraoperative and postoperative care using latest agents so far approved for this purpose.

However the long-term follow up of these patients are needed to conclude firmly regarding the use of newer agents used to reduce Pulmonary Hypertension either intraoperatively are postoperatively.

17 patients among the total patients required a septal approach due to the concomitant presence of tricuspid regurgitation and hence the need for devega procedure done for them.


AGE GROUP
1
2
3
4
5


SEX
1
2


MORTALITY
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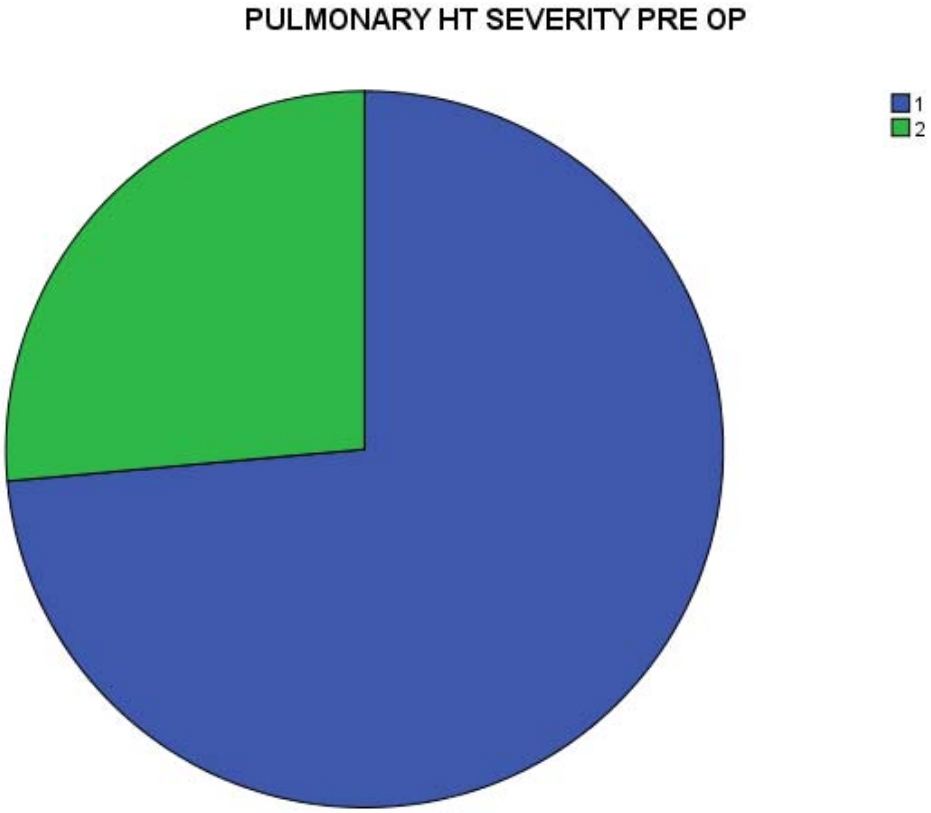
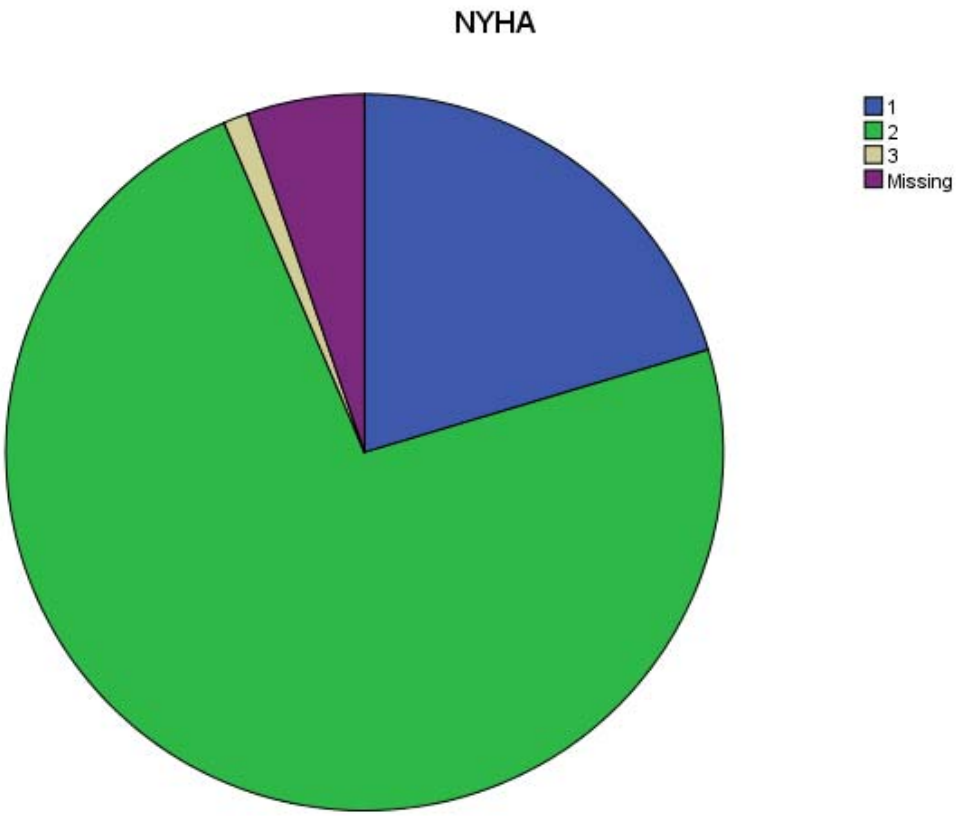
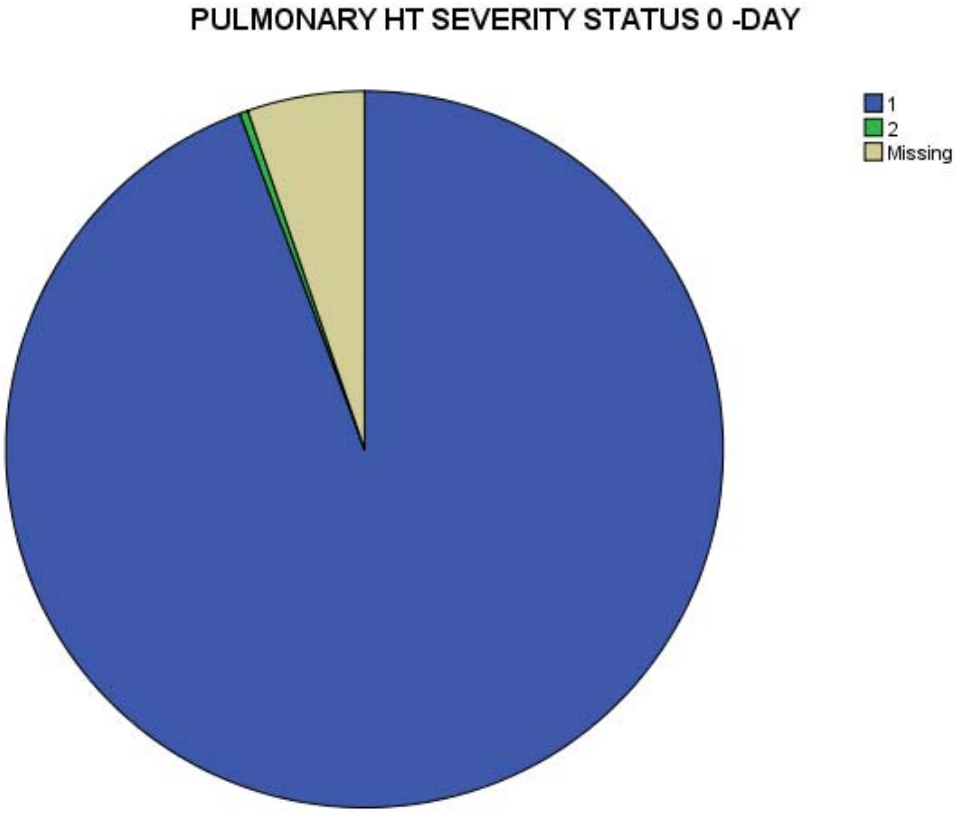
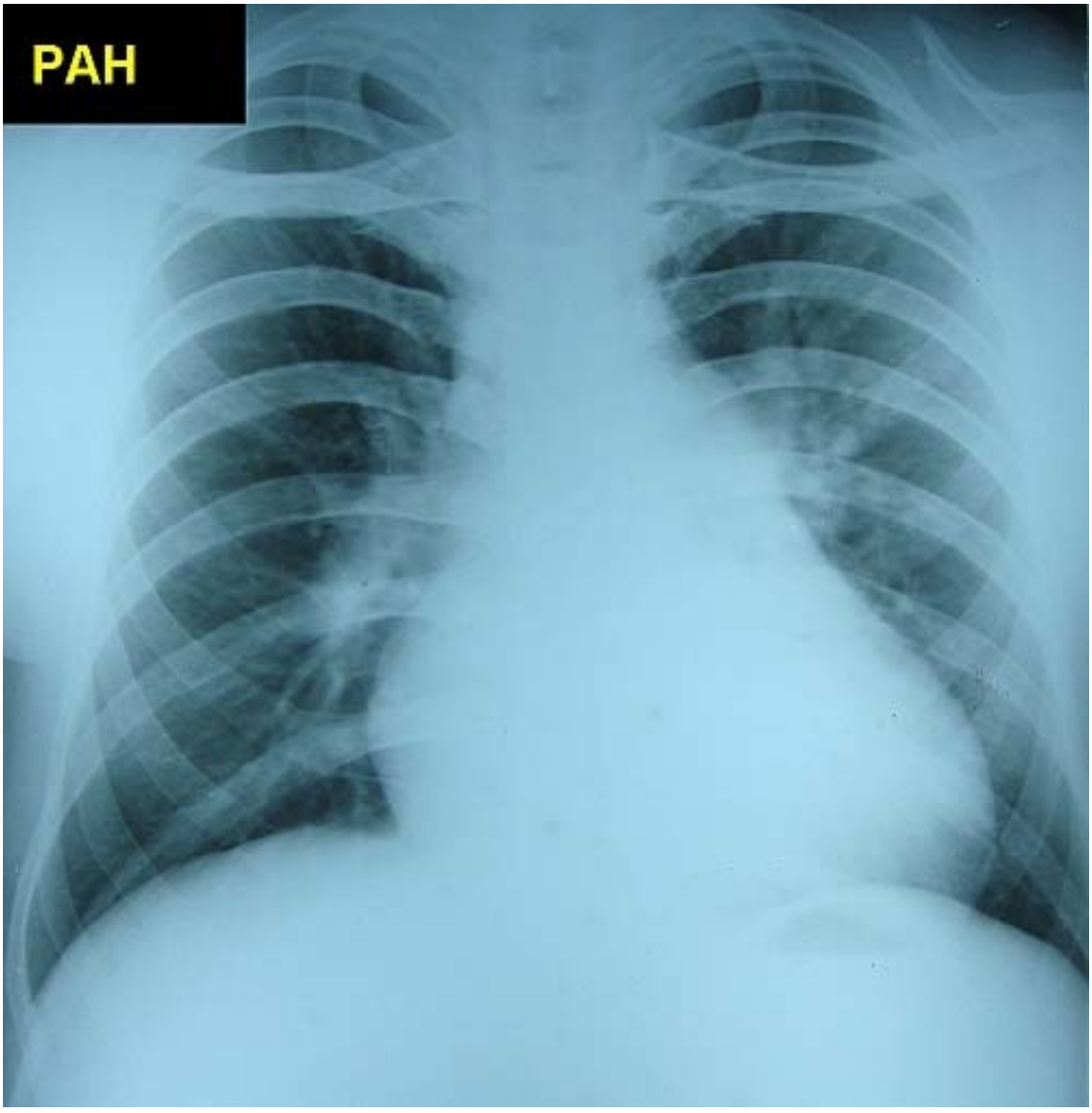
1


PULMONARY HT SEVERITY PRE OP
1
2

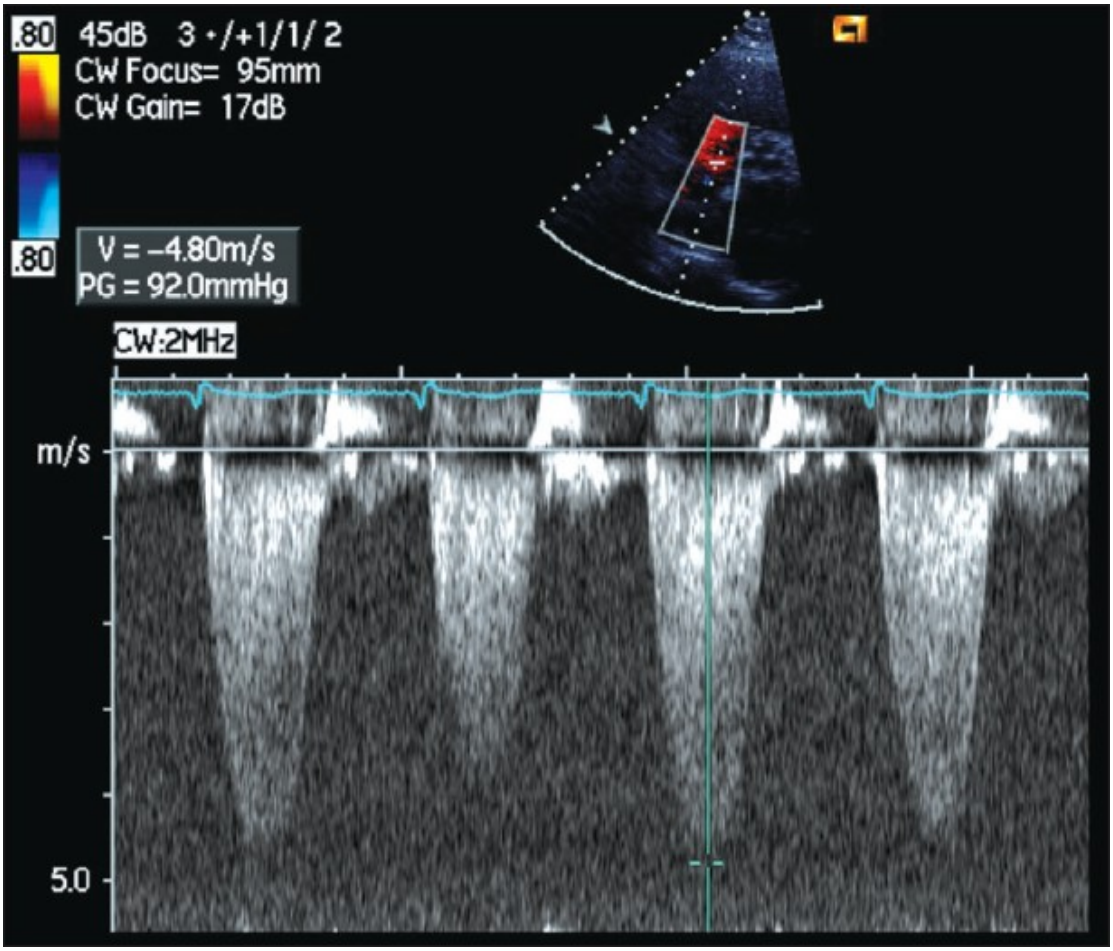

NYHA
1
2
3
Missing


PULMONARY HT SEVERITY STATUS 0 -DAY
1
2
Missing



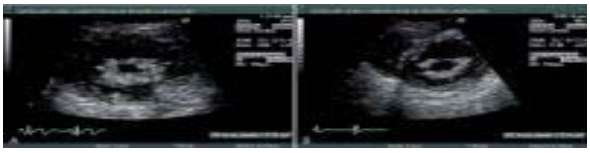
CHEST X-RAY OF SEVERE PULMONARY HYPERTENSION

C

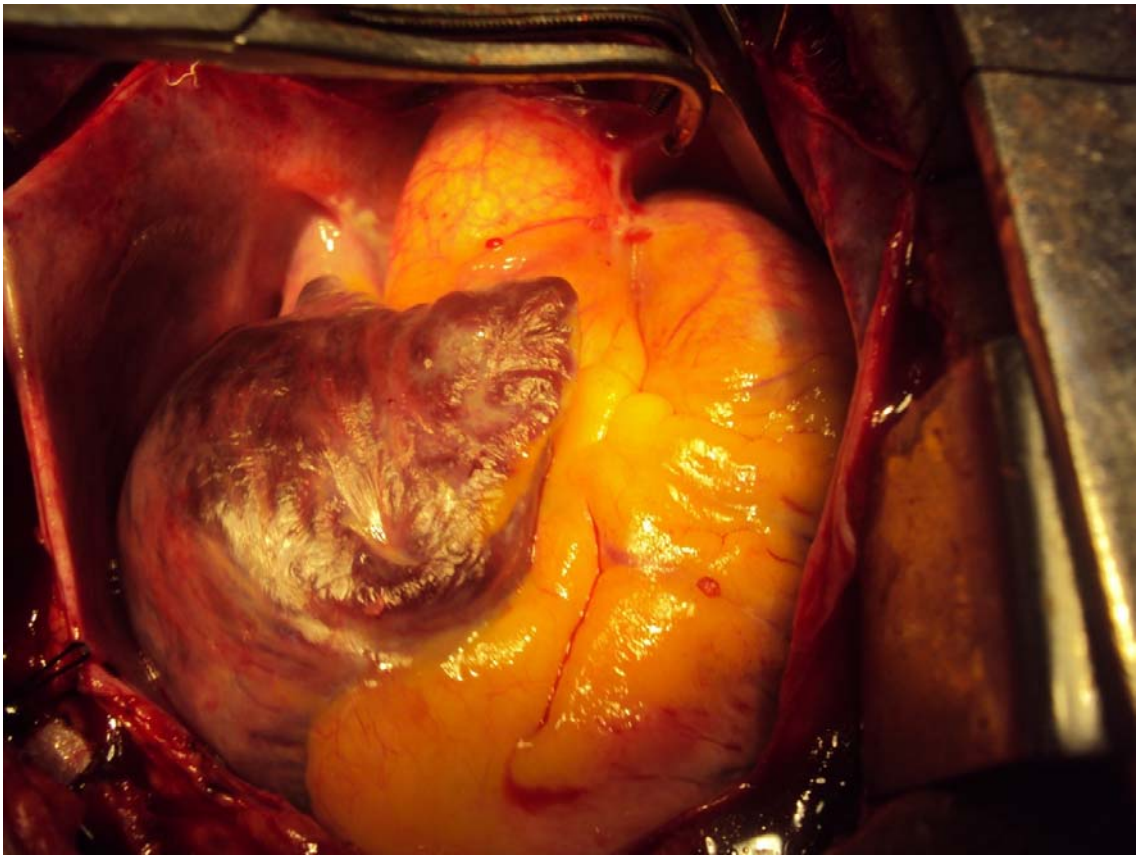


ECHO DOPPLER PICTURE OF SEVERE PULMONARY HYPERTENSION

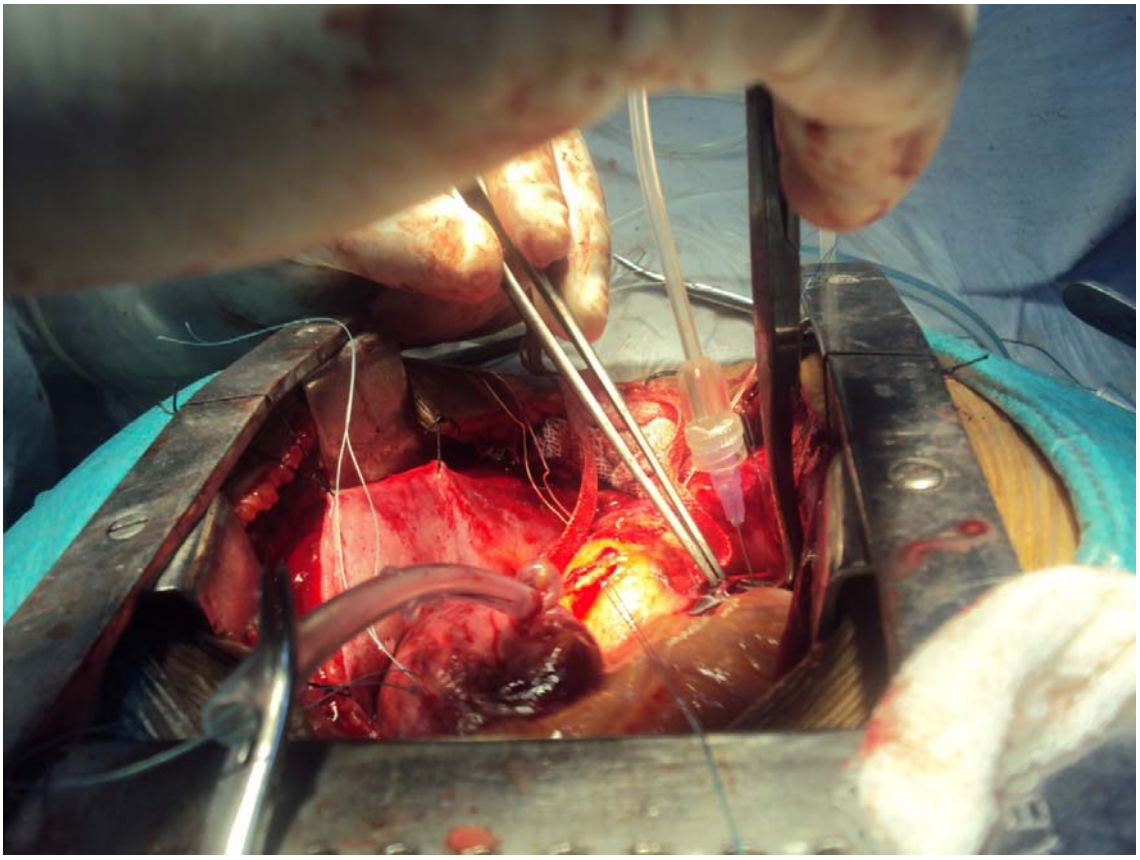
EXCISED SPECIMEN OF MITRAL VALVE



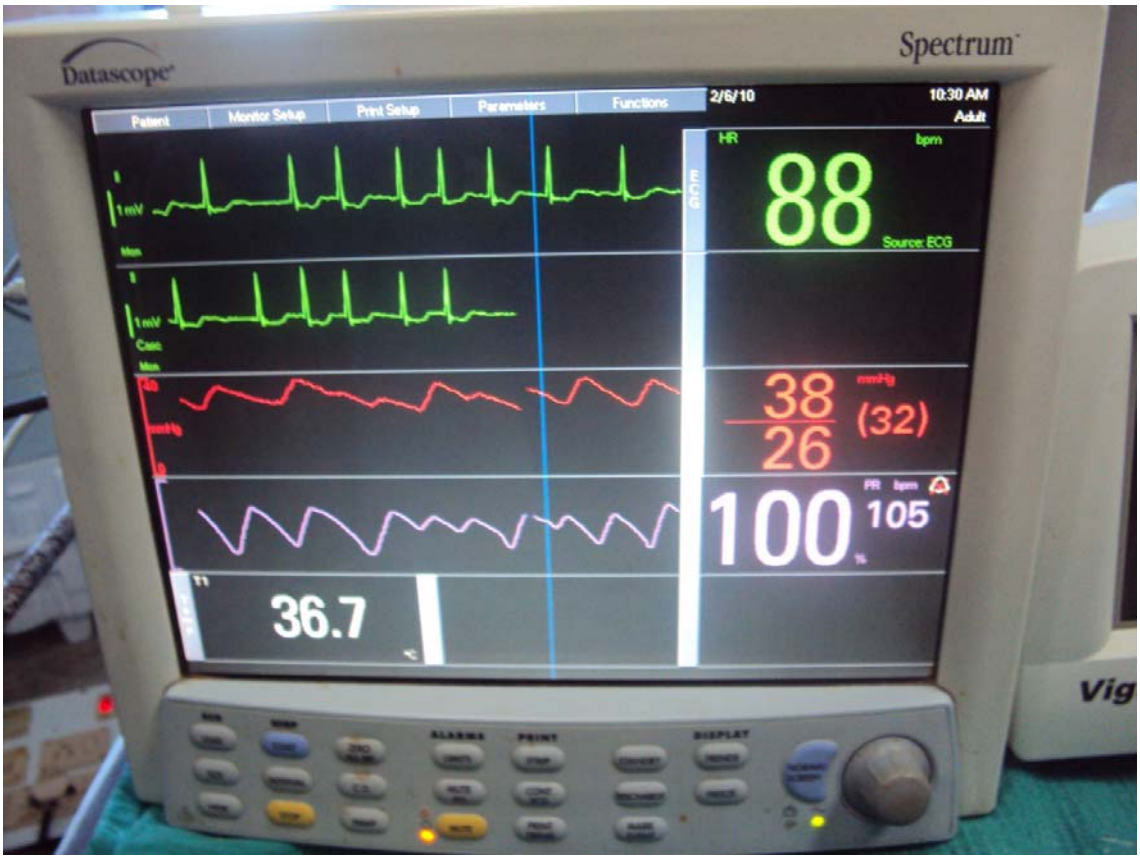
DOPPLER IMAGE OF RHEUMATIC MITRAL STENOSIS



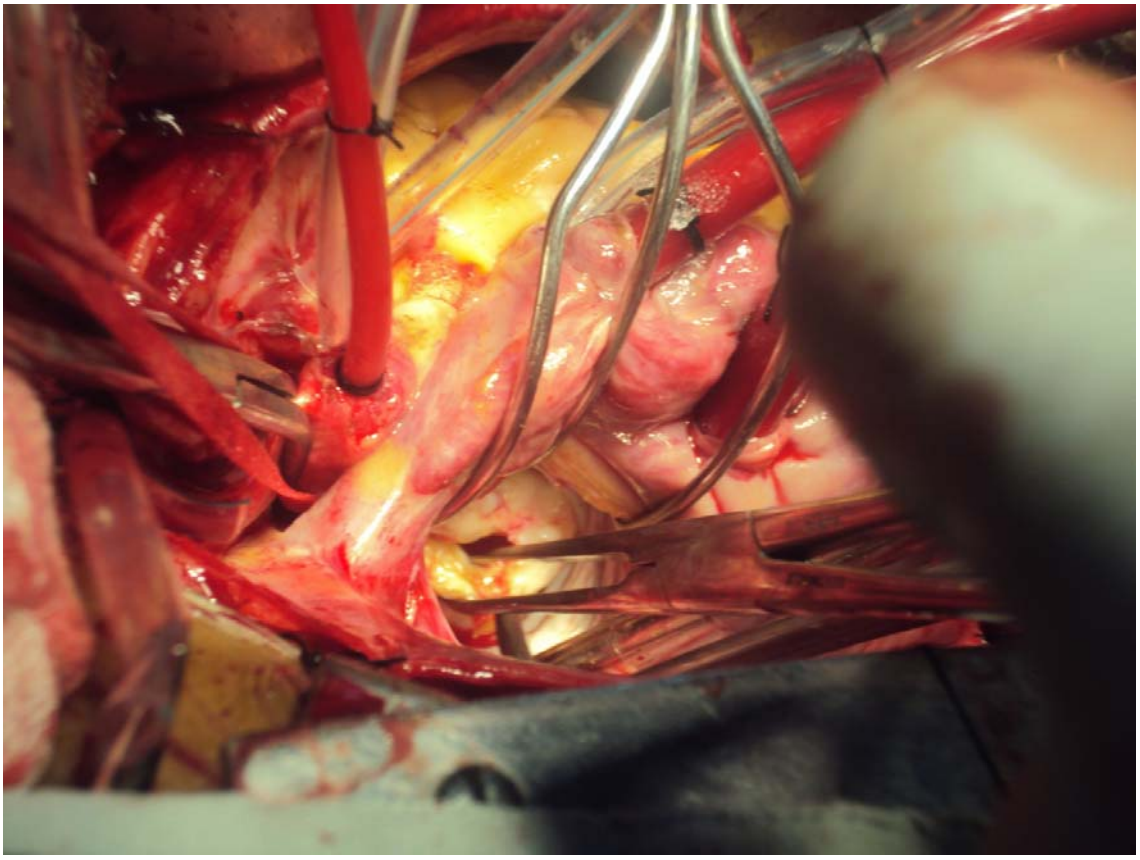
PRE-CARDIOPULMONARY BYPASS PICTURESHOWING DILATED MAIN PULMONARY ARTERY



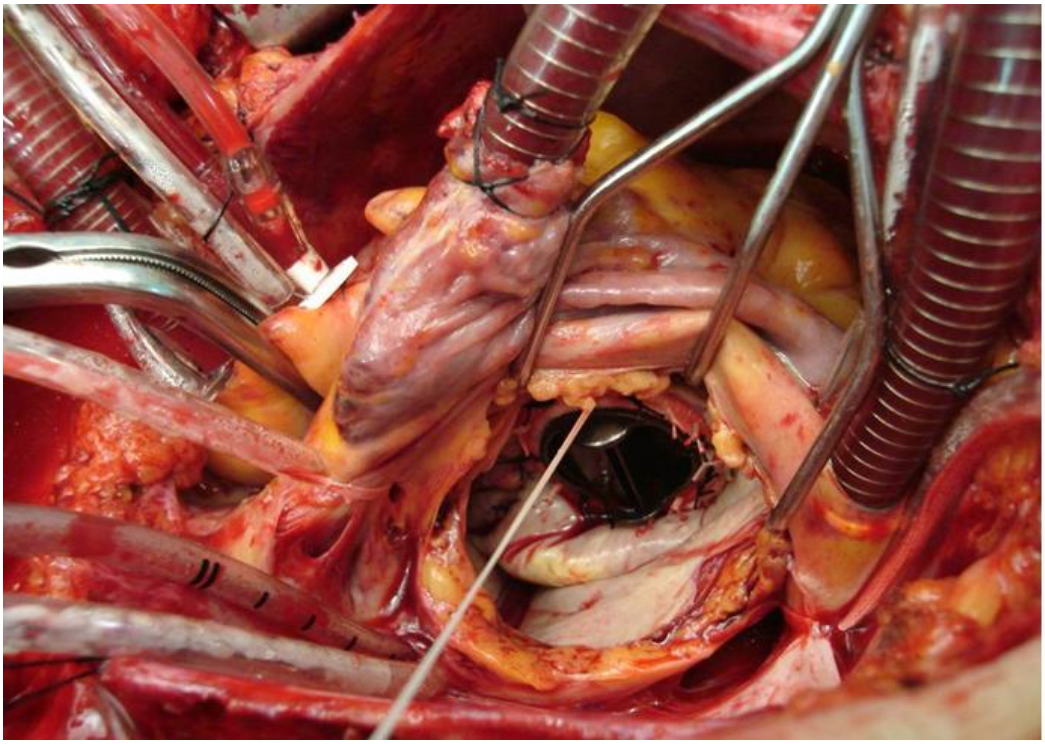
INTRAOPERATIVE PULMONARY PRESSURE MEASUREMENT USING NEEDLE



INTRAOPERATIVE PULMONARY PRESSURE RECORDING



PHOTOGRAPH SHOWING RHEUMATIC MITRAL VALVE EXPOSURE ON CARDIOPULMONARY BYPASS



INTRAOPERATIVE PICTURE SHOWING INSERTION OF St JUDE'S MECHANICAL PROSTHETIC VALVE IN MITRAL POSITION

Summary & Conclusion

SUMMARY &CONCLUSION

Pulmonary arterial hypertension has long been considered a risk factor for poor outcome in patients undergoing MVR, with operative mortality ranging from 15%–31%. Najafi and colleagues found the degree of PAH correlated strongly with perioperative mortality, ranging from 16% in patients with mild PAH to 23% in severe PAH and 61% when PAP was at systemic levels. Recently, several reports have demonstrated improved outcome in patients with PAH undergoing MVR, with perioperative mortality ranging from 2.3%–10%.The improved outcome was attributed to better myocardial preservation, preservation of the subvalvular apparatus, and improved postoperative care. In our study, the overall operative mortality rate was 7.07 % which is consistent with recent reports. However, the mortality rate in patients with PHT overall was 5.5 % which is better than the recent reports.

Numerous studies have examined hemodynamic changes in this subset of patients at different intervals after mitral valve procedures. Most have demonstrated an immediate reduction in PAP and PVR, signifying a sudden drop in left atrial pressure and reversal of the severe spastic Pulmonary vasoconstriction that accompanies left atrial hypertension in some patients. Others have shown slow regression of elevated PAP and PVR several months postoperatively. These reports point toward the involvement of multiple factors in the development of PAH in mitral valve disease. There have been studies of closed mitral commissurotomy and balloon mitral valvotomy in this subgroup of patients from India, which have shown good results in terms of survival, postoperative functional class, and hemodynamics. In our study the mean PAP and PVR did not fall significantly immediately following MVR .The mean fall in PAP was about 46.77 % in males and 45.5% in females which was against the previous views .Although the mean PAP fell significantly from 84.5 to 70.88% mm Hg, it remained within the definition of severe PAH. The PVR showed no significant reduction immediately after MVR, but a gradual regression was seen over a 6 months period and the fall was significant at 6 months period when compared with the preoperative values. This indicates the reactive component of Pulmonary arterial

vasoconstriction, which may be responsible for part of the disproportionate elevation of PVR seen in as many as 20% of patients undergoing mitral valve procedures.

The persistence of residual elevated PAP and PVR well beyond the normal limit in patients suggests an irreversible component of the increased PVR. Other variables that determine the immediate and long-term results of surgery in this subset of patients include advanced age, acute presentation, decreased left ventricular ejection fraction, functional class, right heart failure and increased left ventricular end-diastolic pressure. Vincens and colleagues identified clinical right heart failure as a predictor of operative mortality, and both right ventricular systolic pressure and right ventricular hypertrophy as predictors of poor outcome. Others have identified severe tricuspid regurgitation and the need for concomitant tricuspid surgery as risk factors for operative mortality in this population. In this study, 47% of patients had right ventricular hypertrophy and/or dilatation, and 33% had severe tricuspid regurgitation.

Despite the high operative mortality in most series of MVR in patients with severe PAH, a striking improvement in survival was noted. In our series, functional class improved by one class or more in the majority of survivors. Long-term morbidity was related mainly to anticoagulation and was attributed to poor patient compliance due to illiteracy in this part of the world. Repair of the mitral valve in patients with predominant MR could have avoided these complications but it was not undertaken because of the high rate of repair failure in patients with rheumatic etiology of mitral regurgitation as well as severe subvalvular pathology with calcification of leaflets in most of them.

I acknowledge that the lack of follow-up of Pulmonary vascular dynamics by catheterization constitutes a limitation of this study and was related primarily to economic factors. A postoperative lung biopsy might have added to the information, but this was not undertaken as most of the patients refused consent for it.

I conclude that MVR is safe and effective even in the presence of severe PAH as long as the Pulmonary arterial pressures are below systemic pressures. With supra-systemic PAP, MVR carries a high risk of mortality, and the patient continues to have

persistent PAH in the postoperative period. Significant reduction in PHT following MVR takes place only gradually and in this study about 6 months period showed a decline in PHT as compared to the previous belief of immediate reduction in PHT in majority of the patients.

This explains the multifactorial causes of PHT in patients with Rheumatic mitral valvular heart disease and severe Pulmonary Hypertension.

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Proforma

PROFORMA

NAME: **AGE:** **SEX:**

OCCUPATION: **DATE OF ADMISSION:**

ADDRESS FOR COMMUNICATION:

ADMISSION DIAGNOSIS: MS MS+MR MR LA CLOT

BRIEF HISTORY : DOE ANGINA PALPITATION PND ORTHOPNOEA OLIGURIA PE

NYHA CLASS : 1 2 3 4 5

RHEUMATIC HEART DISEASE : YES NO

H/0 ANTIFAILURE MEDICATION: YES NO

GENERAL EXAMINATION : HT WT HR BP
A /ICTERUS/CYANOSIS/PEDAL OEDEMA/JVP

CVS EXAMINATION : RV IMPULSE LV IMPULSE AF a) present b) absent.

HEART SOUNDS : S1 S2 S1 LOUD VARIABLE SOFT

S3 S4 YES NO

P2 LOUD, SOFT

CARDIAC MURMERS : MDM GRADE

PSM GRADE

RESPIRATORY SYSTEM : BAE ; ADDED SOUNDS
a) RHONCHI b) FINE CREPTS

ABDOMEN : HEPATOMEGALY

SPLENOMEGALY

ASCITES

CNS : NEUROLOGICAL DEFICIT.

INVESTIGATIONS : BLOOD TC DC ESR PCV Hb PL COUNT

BT CT

BLOOD UREA BLOOD SUGAR S. CREATININE S. Na k

ECG RVH P mitrale LVH AF ST-T SEGMENT OTHERS

X-RAY CHEST :

CT RATIO :

BLOOD GROUPING & Rh Typing

ECHO: MVO MVA LEAFLET MOBILITY THICKENING Ca + SCF

LA SIZE **PHT** **a) mild** **b)** **moderate** **c) severe**

LV DIMENSION **EF:**

CORONARY ANGIOGRAM:

INTRA OP DETAILS: TOTAL CPB TIME **TOTAL 'X ' CLAMP TIME** **CP 1 2 3 4**

VALVE SIZE :

PULMONARY ARTERY PRESSURE SYSTOLE: **DIASTOLE:**

POST OP DETAILS: PULMONARY ARTERY PRESSURE SYSTOLE: DIASTOLE:

POSTOP : VENTILATOR SUPPORT : <24 HRS 24-48 HRS 48-72 HRS >72 HRS

INOTROPES : DOP/DOB DOP+DOB DOP/DOB+ADR/ISO OTHERS

COMPLICATIONS : SSSI STERNAL DEHISCENCE LCOS MODS

MORTALITY : LCOS ARRHYTHMIA SUDDEN CARDIAC ARREST

IMMEDIATE POSTOPERATIVE : PULMONARY ARTERY PRESSURE :

3 MONTHS

6 MONTHS

1 YEAR.

MASTER CHART

S.No	NAME	AGE	AGE GROUP*	DURATION SEX	SEX**	DEATH	PREOP PHT (mmHg)	0 MON (mmHg)	3 MON (mmHg)	6 MON (mmHg)	SEVERITY NYHA	NYHA***
1	GANDHIMATHY	45	4	1	1	0	80	40	35	25	1	2
2	MD.ALI	27	2	2	2	0	70	38	32	30	1	2
3	NASRUDEEN	47	4	1	2	0	75	48	30	26	1	2
4	VARALAXMI	42	4	2	1	0	86	52	40	36	1	2
5	ANNATHAI	35	3	2	1	0	78	50	44	30	1	1
6	RAJAMNI	27	2	1	2	0	74	46	40	36	1	2
7	RAJAMAL	29	2	2	1	0	38	30	26	20	1	1
8	KARTHIK	25	2	1	2	0	36	30	24	20	1	1
9	CHANDRA	40	4	2	1	0	88	48	38	30	1	2
10	NADIYA	18	1	2	1	0	78	44	40	36	1	2
11	BALA	30	3	3	2	0	90	54	40	36	1	2
12	MALATHI	20	1	1	1	0	40	32	26	24	1	1
13	RANI	50	5	2	1	0	86	46	40	30	1	2
14	RAMAR	24	2	2	2	0	80	50	40	32	1	2
15	LAKSHMI	45	4	2	1	0	74	46	44	30	1	2
16	PARTHIBAN	27	2	1	2	0	40	32	30	26	1	2
17	MARYRANI	35	3	1	1	0	36	30	28	20	1	2
18	MANJULA	35	3	2	1	0	76	44	40	32	1	2
19	LAKSMIDEVI	45	4	1	1	0	82	46	40	32	1	2
20	MANI	45	4	2	2	0	78	52	46	34	1	2
21	SEKAR	43	4	3	2	0	40	36	30	22	1	2
22	NAGARAJ	48	4	2	2	1	94	88			0	
23	RADHABAI	49	4	1	1	0	76	48	44	34	1	2

24	MANIKANDAN	16	1	3	2	0	40	36	30	28	1	2
25	BALASUBRAMANI 2	27	2	2	2	0	80	56	52	34	1	2
26	RENUKA	20	2	2	1	0	36	30	28	20	1	1
27	SANTHOSH	19	1	2	2	0	68	54	48	40	1	1
28	ELUMALAI	50	5	2	2	0	38	34	30	22	1	2
29	RAMACHANDRA	15	1	2	2	0	88	52	46	38	1	2
30	PONNAMAL	47	4	2	1	0	40	32	30	20	1	2
31	SIVAJIRAO 4	44	4	1	2	0	78	50	46	38	1	2
32	SAROJA 4	49	4	2	1	0	34	30	22	20	1	2
33	UMA	38	3	2	1	0	90	40	34	30	1	2
34	MEENAKSI	28	2	1	1	0	88	38	36	26	1	2
35	ANJALI	30	3	2	1	0	90	48	40	32	1	2
36	THIIRUMATHY	15	1	2	1	0	30	28	24	20	1	2
37	UMAPATHY	45	4	2	2	0	72	56	44	36	1	2
38	INDRANI	34	3	2	1	0	70	44	30	28	1	2
39	SARAVANAN 2	25	2	2	2	0	40	38	36	30	1	1
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43	EZHIL 4	45	4	2	2	0	84	58	50	46	1	2
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52	MURUGAIAN	52	5	2	2	1	80	74				
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63	JEELAN	40	4	2	2	0	36	34	30	20	1	1
64	PETER	50	5	2	2	0	40	34	30	20	1	1
65	SUMATHY	26	2	1	1	0	78	40	34	24	1	2
66	SHANTHI	34	3	2	1	0	76	44	42	30	1	2
67	HARI	45	4	2	2	0	36	30	28	20	1	1
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83	KALAVATHY	44	4	1	1	0	90	48	42	36	1	2
84	SILAMBARASAN	27	2	2	2	0	74	52	50	40	1	2
85	PERUMAYEE	47	4	3	1	0	40	36	30	26	1	1
86	ALGUMANI	42	4	2	2	0	90	48	40	36	1	2
87	SHANU	35	3	2	1	0	78	50	46	24	1	2
88	MUNIAMMAL	42	4	2	1	0	86	48	40	36	1	2
89	ATHIRUBAN 3	39	3	2	2	0	78	44	40	32	1	1
90	NATRAJAN 2	27	2	1	2	0	30	26	20	18	1	1
91	RAJAMMAL	45	4	2	1	0	74	40	36	30	1	1
92	MURUGAN	18	1	2	2	0	78	40	42	32	1	2
93	VENKATESAN	44	4	2	2	0	80	46	38	26	1	2
94	DURAISAMI	43	4	2	2	0	40	38	36	24	1	1
95	GOPI	16	1	1	2	0	80	46	40	38	1	2
96	ELANGOVAN	39	3	2	2	0	74	50	44	34	1	2
97	AYYAPAN	33	3	1	2	0	36	30	28	20	1	1
98	SALEEMA	22	2	1	1	0	40	32	30	24	1	2
99	ELANGOVAN	39	3	3	2	0	76	54	48	34	1	2
100	SAHINBEGUM	46	4	2	1	0	80	48	40	34	1	2
101	SHANTHI	21	2	2	1	0	72	40	36	30	1	2
102	SELVI	18	1	2	1	0	68	40	38	30	1	2
103	RAJI	33	3	2	1	0	80	48	40	32	1	2
104	DHATCHANAMOORTHI	25	2	1	2	0	78	48	44	36	1	2
105	PARTHASARATHI	35	3	2	2	0	36	30	28	22	1	2
106	SEKAR	44	4	2	2	0	80	48	40	38	1	2
107	SHAJIN	46	4	2	1	0	74	48	43	37	1	1
108	SEKAR	44	4	1	2	0	80	58	48	38	1	2

109	SARVANAN	47	4	2	2	0	76	42	38	30	1	2
110	RAJESVARI	14	1	2	1	0	40	35	28	22	1	2
111	DEIVANI	52	5	3	1	0	80	48	47	35	1	2
112	IYYAPAN	20	2	2	2	0	40	30	28	22	1	1
113	ESAKKIAPPAN	40	4	1	2	0	70	48	40	36	1	2
114	PONNAN	45	4	2	2	0	80	58	50	36	1	3
115	LAKSMIDEVI	18	1	2	1	0	70	40	38	33	1	2
116	SUGANTHI	35	3	2	1	0	76	44	39	27	1	2
117	NADIYA	26	2	1	1	0	30	26	20	18	1	1
118	MADAN	15	1	4	2	0	34	30	27	23	1	1
119	ARUN	23	2	2	2	0	40	36	30	22	1	1
120	LALITHA	29	2	2	1	0	78	48	40	42	1	2
121	RANI	28	2	1	1	0	88	40	38	30	1	2
122	SHANMUGAM	42	4	2	2	0	36	32	30	25	1	1
123	JANAKIRAMAN	15	1	1	2	0	86	58	50	32	1	2
124	MAHESVARI	24	2	2	1	0	88	46	40	32	1	2
125	VATCHALA	59	5	2	1	0	86	44	42	38	1	2
126	NANDHINI	30	3	2	1	0	38	34	30	24	1	1
127	GOVINDAN	33	3	2	2	0	88	48	40	37	1	2
128	MARINESAN	53	5	1	2	0	88	49	47	35	1	2
129	VASANTHI	30	3	2	1	0	40	38	30	28	1	2
130	PERIASAMI	49	4	2	2	0	38	32	30	25	1	1
131	KOLANCHIAPPAN	38	3	1	2	0	90	54	47	33	1	2
132	VIDHYA	17	1	2	1	0	44	39	27	20	1	1
133	VASANTHI	25	2	1	1	0	32	30	27	22	1	1
134	DILIP	23	2	2	2	0	80	49	44	38	1	2
135	RAMADURAI	36	3	2	2	0	88	49	42	31	1	2
136	THANGAKODI	61	2	1	1	0	40	38	34	31	1	1
137	GEETHA	31	3	2	1	0	80	48	40	36	1	2
138	AYYASAMI	26	2	2	2	0	80	59	47	34	1	2
139	KESAVAN	37	3	2	2	0	88	53	45	33	1	2
140	MURUGESAN	36	3	1	2	0	90	56	50	37	1	2
141	THENMOZHI	38	3	2	1	0	92	46	44	33	1	2

142	MURUGAMMAL	20	2	2	1	0	90	56	49	37	1	2
143	BASKAR	18	1	2	2	0	88	49	43	37	1	2
144	MINNALKODI	35	3	2	1	0	90	48	42	36	1	2
145	PANDURANGAN	36	3	2	2	0	40	36	30	24	1	1
146	SIVAPRAKASH	40	4	2	2	0	32	28	20	18	1	1
147	DEVI	23	2	1	1	0	86	46	40	38	1	2
148	SIVAGAMI	40	4	2	1	0	40	32	30	24	1	1
149	YASODHA	54	5	2	1	0	86	48	40	34	1	2
150	CHINNARAJ	15	1	2	2	0	46	36	30	22	1	1
151	SARASWATHY 4	35	3	1	1	0	94	48	40	32	1	2
152	RAJA	18	1	2	2	0	90	50	42	36	1	2
153	SHANTHI	23	2	1	1	0	86	40	36	30	1	2
154	MARIAMMAL	29	2	2	1	0	88	48	40	36	1	2
155	SUMITHRA	27	2	1	1	0	90	48	40	36	1	2
156	LAKSHMI	16	1	2	1	1	88					
157	GOPAL	47	4	2	2	0	40	36	30	22	1	2
158	SASIKALA	32	3	2	1	0	36	30	28	22	1	1
159	VIJAYALAKSHMI	14	1	2	1	0	90	46	40	32	1	2
160	PATCHIAPPAN	38	3	2	2	0	88	58	50	40	1	2
161	KOUSALYA	31	3	2	1	0	82	46	43	31	1	2
162	DEVENDRAN	49	4	1	2	0	88	49	41	39	1	2
163	KANNAN	43	4	2	2	0	70	41	37	26	1	1
164	RAMALINGAM	40	4	1	2	0	80	47	40	32	1	2
165	JEYASHANKAR	21	2	2	2	0	78	58	47	32	1	2
166	GAJALAKSHMI	58	5	2	1	0	32	30	27	22	1	1
167	RAMASAMI	40	4	1	2	0	70	55	43	31	1	2
168	KRISNAMOORTHY	49	4	2	2	0	80	44	37	29	1	2
169	THILLAIAMMAL	28	2	2	1	0	78	49	37	27	1	2
170	RAJENDREN	38	3	2	2	0	40	32	20	22	1	2
171	NANDHINI	16	1	2	1	0	86	56	47	32	1	2
172	ROUTRAJ	32	3	2	1	0	70	46	41	28	1	2
173	SATHYA	34	3	2	1	0	38	36	34	30	1	2
174	JEYARAMAN	62	5	3	2	0	82	56	49	37	1	2
175	RAMACHANDRAN	22	2	2	2	0	90	59	44	32	1	2
176	KRISNAMOORTHY	48	4	2	2	0	40	36	32	22	1	2
177	DHANALKSHMI	32	3	2	1	0	90	46	42	36	1	2
178	VIMALA	42	4	2	1	1	90					
179	SAMINATHAN	40	4	1	2	0	76	54	49	32	1	2
180	PARI	32	3	2	2	0	88	48	39	21	1	2
181	VELAYUTHAN	35	3	2	2	0	80	46	40	36	1	2
182	MAALIGA	34	3	2	1	1	90					
183	JEYACHANDRAN	16	1	2	2	0	80	54	49	37	1	2
184	ADAIKALARAJ	24	2	2	2	0	88	48	45	36	1	2
185	MARIAMMAL	17	1	2	1	0	78	46	40	34	1	2
186	RENUKADEVI	20	2	2	1	0	80	47	39	31	1	2
187	SATHYA	18	1	2	1	1	88					
188	RAVI	38	3	1	2	0	90	58	50	42	1	2
189	PALANIAMMAL	37	3	2	1	0	88	48	39	27	1	2
190	LATHA	31	3	2	1	0	80	42	40	27	1	2

191	SARAVANAN 1	14	1	2	2	0	36	30	28	22	1	1
192	BALAMANI	27	2	2	1	0	80	44	40	28	1	2
193	INDRANI	28	2	2	1	0	88	48	36	32	1	2

195	PANNERSELVAM	48	4	1	2	0	90	55	48	37	1	2
196	DHANASELVI	30	3	2	1	0	82	42	40	35	1	2
197	SUDHA	18	1	2	1	0	90	48	40	36	1	2
198	KABILAN	39	3	2	2	0	84	47	38	32	1	2
199	SHEIK	28	2	2	2	0	70	50	40	32	1	2
200	SENNAMA	40	4	3	1	0	38	32	28	22	1	1
201	JEGANNATHAN	35	3	2	2	0	90	57	46	37	1	2
202	TAMILSELVI	36	3	2	1	0	40	36	32	28	1	1
203	PARVATHAM	40	4	2	1	0	86	46	40	32	1	2
204	MUNEESHVARI	35	3	2	1	0	86	46	40	34	1	2
205	MUNUSWAMI	27	2	2	2	0	90	59	47	35	1	2
206	SAMINATHAN	40	4	2	2	0	88	56	47	34	1	2
207	SHANTHI	37	3	1	1	0	38	32	30	24	1	1
208	KOMALA	30	3	1	1	1	40					
209	MURUGESVARI	32	3	2	1	0	80	46	39	32	1	2
210	BALAJI	19	1	2	2	0	78	44	40	32	1	2
211	VENKATESAN	24	2	2	2	0	40	38	33	27	1	1
212	NABISHA	47	4	2	1	0	80	48	39	31	1	2
213	EZHILRANI	28	2	2	1	0	80	56	48	36	1	2
214	KAVITHA	30	3	2	1	0	38	30	28	20	1	1
215	KRISHNAVENI	32	3	2	1	1	90	88				
216	MUTULAXMI	35	3	2	1	1	90	80				
217	VINOTHA	22	2	2	1	1	88	80				
218	RAVIKUMAR	47	4	1	2	0	30	28	26	21	1	1
219	SOUNDARYA	28	2	2	1	0	80	48	42	32	1	2
220	ARJUNAN	16	1	2	2	0	40	36	30	26	1	2
221	DURAISAMI	42	4	2	2	0	88	50	46	31	1	2
222	MALARKODI	31	3	2	1	0	80	42	40	32	1	1
223	KARTHIK	39	3	2	2	0	90	48	36	25	1	2
224	KIRTHIHA	17	1	2	1	0	88	42	40	28	1	2

225	SUDHA	28	2	1	1	1	90	76			
226	VENKATESAN	22	2	2	2	0	78	56	50	35	1
227	PALANI	32	3	2	2	0	40	36	30	32	1
228	PARVATHI	40	4	2	1	0	88	44	40	32	1
229	SELVAMARI	28	2	2	1	0	78	42	40	32	1
230	SUDHA	28	2	2	1	0	40	36	30	22	1
231	SHNTHAKUMARI	40	4	2	1	0	34	32	28	22	1
232	SHANMUGAM	51	5	2	2	0	88				
233	ANJALAIDEVI	26	2	2	1	0	88	50	46	32	1
234	KASTHURI	30	3	2	1	0	78	48	40	32	1
235	KANNIAPPAN	36	3	2	2	0	90	56	50	40	1
236	PALANISAMI	40	4	2	2	0	40	34	32	30	1
237	ABITHA	25	2	1	1	0	90	50	48	32	1
238	GOMATHY	30	3	2	1	0	9048	40	30	26	1
239	JADAMANI	36	3	2	2	0	80	50	44	32	1
240	CHANDRAMATHI	32	3	2	1	0	88	50	47	34	1
241	MUTHU	40	4	2	2	0	80	54	42	36	1
242	SIVAGAMAI	40	4	2	1	0	80	46	42	34	1
243	PRABHU	35	3	2	2	0	90	58	50	32	1
244	SADASIVAM	45	4	1	2	0	78	56	46	32	1
245	EDWARD	58	5	2	2	0	80	48	40	28	1
246	SUGANTHI	17	1	2	1	0	88	48	46	38	1
247	SARALA	23	2	2	1	0	90	42	38	27	1
248	RATHINAVELU	54	5	2	2	0	86	46	40	32	1
249	SARASWATHY 4	23	2	2	1	0	78	46	40	27	1
250	PALANIAMMAL	45	4	2	1	0	70	48	40	34	1
251	SANGEETHA 2	19	1	2	1	0	70	48	40	36	1
252	SNEHA	18	1	2	1	0	90	59	48	31	1
253	SANGEETHA 2	20	2	2	1	0	72	48	40	28	1
254	SUMATHY	35	3	2	1	1	82				
255	SAMUNSEHVARI	15	1	1	1	0	78	48	42	28	1
256	SHANKAR	13	1	1	2	0	80	58	50	45	1
257	MAHESHVARI	35	3	2	1	1	90	80			
258	CHITRA	24	2	2	1	0	90	47	36	28	1
259	KAVITHA	25	2	2	1	0	38	36	32	30	1
260	JANAKI	33	3	2	1	0	80	50	42	34	1
261	LAKSHMI	26	2	2	1	0	40	32	30	22	1
262	MUNUSWAMI	35	3	2	2	0	90	54	48	35	1
263	AYYANAR	35	3	2	2	0	86	54	50	40	1
264	GOPALAKRISHNAN	42	4	1	2	0	86	52	48	32	1
265	VIJAYA	37	3	2	1	0	80	56	48	32	1

* Age Group
1- 10-19 years
2- 20-29 years
3-30-39 years
4- 40-49 years
5-> 50 years

** Sex
1- Female
2-Male

***NYHA
1- Class I
2-Class II
3-Class III

CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY TO ANALYSE PREOPERATIVE AND POSTOPERATIVE SEVERE PULMONARY HYPERTENSION FOLLOWING MITRAL VALVE REPLACEMENT FOR MITRAL VALVULAR HEART DISEASE"** is the bonafide original work of **DR. A. ARUNKUMAR** in partial fulfillment of the requirements for M.Ch. Branch-I CARDIO-VASCULAR & THORACIC SURGERY examination of THE TAMIL NADU DR. M.G.R MEDICAL UNIVERSITY to be held in August 2010. The period of post-graduate study and training was from August 2007 to July 2010.

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DECLARATION

I **Dr. A. ARUNKUMAR,** solemnly declare that this dissertation entitled, **"A STUDY TO ANALYSE PREOPERATIVE AND POSTOPERATIVE SEVERE PULMONARY HYPERTENSION FOLLOWING MITRAL VALVE REPLACEMENT FOR MITRAL VALVULAR HEART DISEASE"** is a bonafide work done by me at the Department of Cardio Vascular & Thoracic Surgery, Madras Medical College and Government General Hospital during the period 2007 – 2010 under the guidance and supervision of the Professor and Head of the Department of Cardiothoracic Surgery, Madras Medical College and Government General Hospital, **Prof. S. Manoharan, M.S., M.Ch.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.Ch. Degree (Branch – I) in Cardio-vascular & Thoracic Surgery.**

Place : Chennai

Date: 24th May 2010

Dr. A. ARUNKUMAR

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Contents

INTRODUCTION

Rheumatic Heart disease has been one of the major health problems in developing countries like India. This is an autoimmune disease which occurs in the cardiac tissues due to streptococcal throat infection. Pancarditis and particularly vasculitis contributes the major complication following this disease. Mitral valve constitutes about 50 % of the valve which gets affected due to rheumatic heart disease. Pulmonary Hypertension is said to complicate about 70% of the patients affected by this disease. Pulmonary Hypertension adversely affects the prognosis and course of the disease.

Pulmonary Hypertension in mitral valvular heart disease leads to various adverse outcome following surgical treatment of this condition. In majority of the patients this Pulmonary Hypertension is reversible following surgery. In considerable number of patients however the persistence of Pulmonary Hypertension leads to postoperative problems which may result in death.

Understanding the Pathophysiology of the Pulmonary Hypertension occurring due to this disease and analyzing it before embarking on the treatment option would largely benefit the team involved in the care of suffering patients.

Management of Pulmonary Hypertension before surgery involves investigations and manipulation with certain drugs which may reduce the Pulmonary pressures before proceeding towards surgical correction.

An attempt of a prospective study to analyze the facts is made and the results are tabulated and compared to the national and international views on the same parameter of this common disease.

Pulmonary Hypertension is diagnosed clinically by identifying loud P2 component of 2nd Heart sound, TR Murmur and the accompanying ECG changes

like Right Atrial enlargement, Right Axis deviation and electrical rotation of the Heart.

Pulmonary Hypertension is readily identified by Echocardiography which is also used to classify the pressures into mild, moderate and severe categories. Catheterization studies are not done routinely as echocardiogram has emerged as an effective tool to identify Pulmonary Hypertension and classify accordingly.

In our study which was conducted for a period of 2 years from July 2007 to July 2009 we had recorded data of about 265 patients with Mitral Stenosis or Mitral Regurgitation cases with severe Pulmonary Hypertension. We had investigated them with preoperative ECHO and intraoperative PAP measurement using transducers and followed them with postoperative Echocardiogram for about 6 months.

All patients were well informed about the procedure and appropriate consent had been obtained prior to the valve replacement.

The results obtained from the data collection were tabulated and analyzed using various parameters which may affect the outcome of the patients and were statistically scrutinized using appropriate software.

Review of Literature

REVIEW OF LITERATURE

PULMONARY HYPERTENSION -MECHANISM

Patients with Rheumatic Heart Disease develop Pulmonary Hypertension due to various reasons and prime cause among them will be the retrograde transmission of LA Hypertension which gets transmitted to the Pulmonary arteries. Pulmonary venous pressure also is transmitted to Pulmonary arteries. There is also reactive Pulmonary Arteriolar constriction which can lead to Pulmonary Hypertension. For some unknown reasons there are morphological changes in Pulmonary Vasculature which can lead to the development of Pulmonary Hypertension. Interstitial Oedema which can occur due to elevated LA pressure >20 mmHg can also lead to the development of Pulmonary Hypertension.

Pathophysiology of Pulmonary Hypertension

Elevated LA pressure

⇩

Elevated Pulmonary venous pressure

⇩

Transmitted pulmonary venous hypertension to arteries

⇩

Vasoconstriction of Pulmonary arterioles

⇩

Pulmonary Artery Hypertension

⇩

Right ventricular dilatation

⇩

Tricuspid Regurgitation

⇩

Right Atrial enlargement

⇩

Congestive cardiac failure

The above flow chart explains the Pathophysiology of Pulmonary Hypertension in Mitral Valvular heart disease in both Rheumatic Mitral Stenosis and Regurgitation.

Pulmonary Hypertension greatly influences the natural course of the disease process, treatment response and also the post intervention prognosis.

Wood et al had noted that severe PHT was associated with both moderate MS and severe MS. Fawzy and Reibero pointed out that there is another group of patients with severe MS, yet only a mild increase in PAP. The reasons for not developing PHT in these cases are not clear. No significant relation between LA size and PAP has been noted. There is a view that the increase in LA size would cushion of the sequelae of increase in pressure.

Bahl et al and Krishnamoorthy et al had observed that PHT is an indicator of disease severity in patients with Mitral Stenosis. Chronicity of the disease process as indicated by the presence of severe fibrosis and Atrial Fibrillation are important in the development of reactive PHT

FEATURES INDICATING SEVERITY OF DISEASE IN MITRAL VALVULAR LESIONS COMPLICATED BY PULMONARY HYPERTENSION

- Severe Subvalvular Pathology
- Small Mitral Valve Area
- Higher Transvalvular gradient
- Higher Pulmonary valve resistance
- Higher NYHA symptoms
- Thickened non pliable valves
- Higher Wilkins score
- Higher incidence of Atrial Fibrillation.

DEFINITION:

In medicine, Pulmonary Hypertension (PH or PHT) is an increase in blood pressure in the Pulmonary artery, Pulmonary vein, or Pulmonary capillaries, together known as the Lung Vasculature, leading to shortness of breath, dizziness, fainting, and other symptoms, all of which are exacerbated by exertion. Pulmonary Hypertension can be a severe disease with a markedly decreased exercise tolerance and heart failure. It was first identified by Dr. Ernst von Romberg in 1891

The Venice 2003 revised classification system can be summarized as follows[4]

WHO Group I - Pulmonary Arterial hypertension (PAH)

Idiopathic (IPAH)

Familial (FPAH)

Associated with other diseases (APAH): Collagen Vascular Disease (e.g. Scleroderma), congenital shunts between the systemic and Pulmonary Circulation, portal hypertension, HIV infection, drugs, toxins, or other diseases or disorders

Associated with venous or capillary disease

WHO Group II - Pulmonary Hypertension associated with left heart disease

Atrial or Ventricular disease

Valvular disease (e.g. Mitral Stenosis)

WHO Group III - Pulmonary Hypertension associated with lung diseases and/or Hypoxemia

Chronic Obstructive Pulmonary disease (COPD), Interstitial Lung Disease (ILD)

Sleep-disordered breathing, Alveolar Hypoventilation

Chronic exposure to high altitude

Developmental lung abnormalities

WHO Group IV - Pulmonary Hypertension due to chronic thrombotic and/or embolic disease

Pulmonary Embolism in the proximal or Distal Pulmonary Arteries

Embolization of other matter, such as tumor cells or parasites

WHO Group V - Miscellaneous

This Classification does not include sickle cell disease, Human Herpesvirus 8, also associated with Kaposi's sarcoma, and has been demonstrated in patients with PAH, suggesting that this virus may play a role in its development. Recent studies have been unable to find an association between human Herpesvirus 8 and Idiopathic Pulmonary Arterial Hypertension.

Pulmonary Hypertension – Heath Edwards grading system

Grade	**Microscopic Features**
	Potentially Reversible
I	Hypertrophy of the media of muscular Pulmonary Arteries. Extension of muscle into the wall of Pulmonary Arterioles.
II	Muscle Hypertrophy plus proliferation of intimal cells in arterioles and small muscular arteries.
III	"Muscle hypertrophy plus sub endothelial fibrosis. Eventually, concentric masses of fibrous tissue and reduplicated internal elastic lamina occlude the vascular lumen of arterioles and small muscular arteries. Large elastic arteries show atherosclerosis."

Grade	Microscopic Features
	Usually Irreversible
IV	"Muscle hypertrophy is less apparent; progressive dilatation of small arteries, especially those near vessels with Intimal fibrous occlusion. Plexiform lesions occur."
V	Plexiform and Angiomatoid lesions plus intra-alveolar Hemosiderin-filled macrophages.
VI	Necrotizing Arteritis with thrombosis. Fibrinoid necrosis of the arterial wall with a Transmural infiltrate of Polymorphonuclear leukocytes and Eosinophils.

Usually Pulmonary Hypertension grade 3 and below are reversible and can be taken up for surgery with better results. Pulmonary Hypertension grade 4 and above has significant contribution for postoperative morbidity and mortality.

PATHOLOGICAL FINDINGS:

Pathological changes in lungs in rheumatic MS include prominent vascular and parenchymal changes. Pulmonary veins develop muscular media. Moderate to marked medial hypertrophy occurs in medium sized branches of the Pulmonary arteries. Dilatation lesions and plexiform lesions are rarely seen in rheumatic MS .Tandon et al and Chopra et al has reported such lesion in 4% of the autopsy studies conducted in New Delhi, India. Mubeen et al, recently has reported that Pulmonary vascular changes do not go beyond grade 3 (Heath Edward) criteria in RHD. The most striking feature seen is prominent smooth muscle layer in Bronchoalveolar walls. The extent and severity of both vascular and parenchymal changes are seen more in juvenile MS. Haemosiderosis has been reported in long standing cases.

TREATMENT OPTIONS:

- MEDICAL
- INTERVENTIONAL
- SURGICAL

MEDICAL:

Patients with Mitral Valvular heart disease and PHT are started with rate controlling drugs and diuretics once they become symptomatic. Rate controlling drugs like Beta-blocker and Calcium channel blockers are started to control AF which otherwise would worsen the condition. Rate control with these drugs help in reducing the Transmitral gradient and hence the PHT. Digoxin is usually started for the failing heart and RV dysfunction which has to pump blood against severe Pulmonary Hypertension.

INTERVENTIONAL:

Isolated MS and PHT were treated by closed Mitral Commisurotmy (CMC) and Balloon Mitral Valvotomy (BMV) from time immemorial. Balloon Mitral Valvotomy can be attempted only if the valves are pliable and the Wilkins score is favorable. There are many concerns before proceeding to BMV like the less tolerance to the stress of the procedure, difficulty in negotiating the septum in large right sided chambers, Tight Mitral Stenosis and fear of tearing the mitral valve and creating Mitral Regurgitation. However the AHA GUIDELINES 2008 clearly states the role of BMV in a case of severe PHT associated with rheumatic heart disease. Even though BMV has given good results in MS and PHT the mitral valve area remained less on comparison with open procedures with reasonable reduction in Pulmonary vascular resistance and PAP.

SURGICAL:

Patient's hemodynamic performance was better following reduction in Pulmonary vascular resistance particularly after MVR and valvotomy. Pulmonary Hypertension regresses after the transmitral gradient reduced following surgery. Hemodynamic studies done in patients who had undergone MVR showed a reversibility of Pulmonary Artery Hypertension[19]. Various options are open for surgical management of this condition like Mitral valve replacement (MVR), Closed Mitral Commisurotmy and Open Mitral Valvotomy. Mitral Valve Replacement may be done with either mechanical prosthetic valve or Bioprosthetic Valve Autologous transfer of Pulmonary valve (ROSS II PROCEDURE) has also been done for this condition.

Cesjnvar[8] et al has reported higher early mortality among his series of 382 patients who underwent MVR for Mitral Valvular disease with Pulmonary Hypertension. But the late mortality was no different among patients with or without Pulmonary Hypertension. Aris[9] et al has confirmed this finding with his series of 88 patients. Perioperative mortality with patients having Suprasystemic Pulmonary Arterial pressures were 5 times more than with normal or sub systemic pressures.

In view of this fact subjecting patients with Mitral Valvular heart disease for earlier surgical intervention would ameliorate the condition before developing severe Pulmonary Hypertension.

PERSISTENT PHT FOLLOWING MVR

Following MVR majority of the patients have regression of PAP, yet Pulmonary Vascular hypertension remains unchanged in significant proportion of patients[19]. Patients with Suprasystemic PAP continue to have persistently elevated PVR. Patients with irreversible PHT after MVR are found to have early mortality. Compared to patients who have AF, regaining sinus rhythm after MVR results in reduction in PAP.

Patient –prosthetic mismatch contributes significantly to the persistence of Pulmonary Hypertension after MVR. Recent studies had demonstrated the correlation between

persistent PHT and patient –prosthesis mismatch and associated poor prognosis. Patient –prosthesis mismatch occurs with both mechanical and Bioprosthesis Valves.

TRICUSPID REGURGITATION AND PHT

Functional TR occurs following severe PHT in a Rheumatic Mitral Valvular heart disease. There are conflicting reports regarding the resolution of TR following Mitral Valve Replacement. Persistence of TR may contribute to the mortality and morbidity following surgeries done for mitral valve disease. Some times simultaneous TR repair is recommended during surgeries done for mitral valve disease. Patients with severe PHT show regression of TR following MVR and other surgeries for mitral valve disease.

PERIOPERATIVE MANAGEMENT OF PHT PATIENTS

Perioperative mortality is higher with patients having severe PHT and mitral valvular heart disease. But late survival curves are similar for patients with or without Pulmonary Hypertension, therefore effective perioperative management of severe Pulmonary Hypertension would help in the late survival of these patients and there would be no significant difference in the Kaplan-Meier survival curves.

Various drugs are available for managing these patients perioperatively like milrinone, nesiridite, inhaled nitric oxide and prostacyclin and on the horizon we also have sildanefil and endothelin antagonist like bosentan etc which has been used both preoperatively and postoperatively .

Role of phenoxybenzamine has been found be useful in cases of Pulmonary Hypertension secondary to congenital heart disease and in the peri and postoperative periods of congenital heart disease correction especially with elevated Pulmonary vascular resistance .

TREATMENT TARGETED AT PULMONARY VASCULATURE

So far traditionally treatment for mitral valvular heart disease focused only on cardiac physiology. Now the recent concepts are focusing more in the Pulmonary Vascular physiology following mitral valve disease and are due to the renewed interest in Cardio Pulmonary Hemodynamics. Madden[15] et al has recently found the role of phosphodiesterase 5 inhibitor (sildenafil) in a study conducted by him and found to have a positive role in treating these patients. The therapy has been found to be significantly tolerated and results are good as early as 8 weeks .Even exercise tolerance seems to be improved with this medicine.

PULMONARY VASCULAR RESISTANCE

Pulmonary vascular resistance has two components an organic element and dynamic element. Dynamic component is relieved immediately following reduction of LA pressures. Organic element due to changes in Pulmonary vascular changes regresses immediately or may take a long time to do. Kaul and colleagues reported on 30 patients with severe Pulmonary Hypertension which showed striking regression from a mean of 74mm Hg. Restudy an average of 5.5 years after MVR showed an average systolic pressure of 48mmHg and mean of 31 mmHg. This drop was largely due to sudden reduction of LA pressure and reversal of severe spastic Pulmonary vasoconstriction that accompanies left Atrial Hypertension. These changes in Pulmonary vascular resistance are a progressive regression and are the same for both stenosis and regurgitation. In older patients and in patients with AF the regression occurs less frequently.

CALCULATION OF PULMONARY ARTERY PRESSURE

Using Doppler we can measure the Pulmonary artery pressure using the peak Transtricuspid flow velocity (VMAX).

PAP = $4V\ max^2$ + RIGHT ATRIAL PRESSURE.

RAP is usually equal to jugular venous pressure measured clinically or from the Inferior Vena Cava diameter measured in Expiration and percentage collapse of IVC in Inspiration .

MITRAL STENOSIS

The mitral valve is made up of the annulus, anterior and posterior leaflets, and chordae, which attach the leaflets to their respective papillary muscles. A normally functioning valve allows blood to flow unimpeded from the left atrium to the left ventricle during diastole and prevents regurgitation during systole. Normal mitral valve function is dependent not only on the integrity of the underlying valvular structure, but on that of the adjacent myocardium as well.

Definition and Causes

Mitral stenosis (MS) refers to narrowing of the mitral valve orifice, resulting in impedance of filling of the left ventricle in diastole. It is usually caused by rheumatic heart disease. Less common causes include severe calcification of the mitral annulus, infective endocarditis, systemic lupus erythematosus, rheumatoid arthritis, and carcinoid heart disease.

Pathophysiology and Natural History

Patients with MS typically present more than 20 years after an episode of rheumatic fever. Single or recurrent bouts of Rheumatic Carditis cause progressive thickening, scarring, and calcification of the mitral leaflets and chordae. Fusion of the commissures and chordae decreases the size of the mitral opening. This obstruction results in the development of a pressure gradient across the valve in diastole and causes an elevation in left atrial and Pulmonary venous pressures. Elevated left atrial pressures lead to left atrial enlargement, predisposing the patient to Atrial Fibrillation and Arterial Thromboembolism. Elevated Pulmonary Venous pressure results in

Pulmonary Congestion and Pulmonary Edema. In advanced mitral stenosis, patients develop Pulmonary Hypertension and right-sided heart failure.

Signs and Symptoms

Patients with mitral stenosis may present with Exertional Dyspnea, fatigue, atrial arrhythmias, embolic events, angina-like chest pain, Hemoptysis, or even right-sided heart failure. Previously asymptomatic or stable patients may decompensate acutely during exercise, emotional stress, pregnancy, infection, or with uncontrolled atrial fibrillation.

The characteristic findings of MS on auscultation are an accentuated first heart sound, an opening snap, and a mid-diastolic rumble. The first heart sound may be diminished in intensity if the valve is heavily calcified, with limited mobility. If the patient is in sinus rhythm, there is presystolic accentuation of the murmur during atrial contraction. With increasingly severe stenosis, the duration of the murmur increases and the opening snap occurs earlier during diastole as a result of higher left atrial pressure. There is accentuation of P_2 when Pulmonary Hypertension is present. If flow across the mitral valve is reduced because of heart failure, Pulmonary Hypertension, or aortic stenosis the murmur of mitral stenosis may be reduced in intensity or may be inaudible.

Left Atrial Myxoma may be distinguished from MS by the presence of a “tumor plop” versus an opening snap in early diastole.

Diagnosis

On chest radiography, the characteristic findings of mitral stenosis are pulmonary congestion, enlargement of the main Pulmonary arteries, and enlargement of the left atrium without cardiomegaly. An electrocardiogram (ECG) may reveal evidence of left atrial enlargement, Atrial Fibrillation or, in advanced disease, right ventricular hypertrophy consistent with Pulmonary Hypertension.

Two-dimensional (2D) and Doppler echocardiography is indicated for all patients with suspected MS to confirm the diagnosis and determine its severity (Class I indication). Characteristic findings of MS include valve thickening, restricted valve opening, anterior leaflet doming, and fusion of the leaflets at the commissures. The mean pressure gradient across the mitral valve on Doppler echocardiography (echo) in MS is at least 5 mm Hg; in severe stenosis, it is usually higher than 10 mm Hg. Because the gradient across the mitral valve is flow dependent, the severity of MS is more accurately defined by the mitral valve area (MVA). The normal valve area is 4 to 5 cm^2. In mild mitral stenosis, the MVA is 1.5 to 2 cm^2, in moderate stenosis it is 1 to 1.5 cm^2, and in severe stenosis it is less than 1 cm^2. The valve area may be measured by tracing the mitral valve opening in cross section by 2D echo. Alternatively, the MVA is calculated using the pressure half-time (P ×-½t), which is the amount of time it takes for the transmitral pressure to fall to one half its initial value (MVA = 220/[P ×- ½t]).

Echocardiography also allows assessment of Pulmonary artery pressures, detection of other valve disease, visualization of left atrial thrombus, and identification of important differential diagnoses, such as Left Atrial Myxoma. Tran esophageal echo is superior to transthoracic echo at identifying left atrial thrombus in patients who are being considered for Percutaneous Mitral Balloon Valvotomy or Cardio Version. Stress echocardiography may be helpful if there is a discrepancy between a patient's severity of symptoms and the baseline hemodynamic data. An exercise mean transmitral gradient of more than 15 mm Hg and peak right ventricular systolic pressure of more than 60 mm Hg indicate hemodynamically significant MS.

Cardiac catheterization is not necessary in all cases but, like stress echocardiography, may be helpful in characterizing the severity of mitral stenosis when there is a discrepancy between symptoms and findings on echocardiography.

Treatment

Medical Treatment

Medical therapy has no role in altering the natural history or delaying the need for surgery in patients with MS. Medical treatment is directed toward alleviating Pulmonary Congestion with diuretics, treating Atrial Fibrillation, and anticoagulating patients who are at increased risk of arterial embolic events.

Development of Atrial Fibrillation frequently leads to an acute deterioration in patients with mitral stenosis. The rapid ventricular response results in a decrease in the diastolic filling time. Beta blockers, calcium channel blockers, or digoxin may be used to control ventricular rate. An attempt to restore sinus rhythm with direct current electrical cardioversion or antiarrhythmic drugs may be considered. Anticoagulation with warfarin is indicated to prevent thromboembolism when Atrial Fibrillation is present, if there is a prior history of thromboembolism, or a thrombus is detected in the left atrium (Class I). Although controversial, anticoagulation may also be considered if the left atrium is markedly dilated (5.0 to 5.5 mm) or if there is spontaneous contrast on echocardiography (Class II b).

Antibiotic therapy is important for the secondary prevention of Rheumatic Carditis. Patients with a history of rheumatic fever are at high risk of recurrence. Long-term secondary prophylaxis, preferentially with penicillin, is therefore recommended for all patients with a history of rheumatic fever or suspected rheumatic valve disease. The duration of prophylaxis depends on a number of factors, including the time lapsed since the last attack, the age of the patient, the presence or absence of cardiac involvement, and the patient's risk of exposure to streptococcal infections. Routine antibiotic prophylaxis for endocarditis is no longer recommended for patients with mitral stenosis.

Surgery

Three invasive options are available for patients with MS: (1) Percutaneous Mitral Balloon Valvotomy (PMBV); (2) Surgical Mitral Commissurotomy; and (3) Mitral Valve Replacement (MVR). In experienced centers, PMBV is the initial procedure of choice and should be considered for (1) symptomatic patients (NYHA functional Classes II to IV) with moderate or severe MS (Class I) and (2) asymptomatic patients with moderate or severe MS and Pulmonary Hypertension (Class I). PMBV is a catheter-based technique in which a balloon is inflated across the stenotic valve to split the fused commissures and increase the valve area. The MVA typically doubles in size, and hemodynamic as well as clinical improvements are seen immediately. The results are comparable with those achieved with open Mitral Commissurotomy, but it is less invasive and less costly. The Mitral Valve Morphology is an important predictor of successful balloon valvotomy. Severe valve calcification or significant involvement of the subvalvular apparatus on echocardiography before PMBV is associated with a higher complication rate and a greater risk of recurrence. In addition, balloon valvotomy should not be performed in patients who have left atrial thrombus or more than 2+ (moderate) mitral regurgitation, because the degree of mitral regurgitation usually increases following the procedure. Complications of Balloon Mitral Valvotomy include severe Mitral Regurgitation (3%), Thromboembolism (3%), and residual Atrial Septal Defect with significant shunting (less than 5%). Mortality with the procedure is lower than 1% in experienced hands. At 7 years after balloon valvotomy, 50% to 69% of patients remain free of cardiovascular events and up to 90% of patients remain free of reintervention. However, both Balloon Valvotomy and Surgical Commissurotomy are palliative procedures and, in most cases, further intervention is eventually required, usually in the form of a Mitral Valve Replacement.

In patients with calcified valves that cannot be treated by valvotomy or commissurotomy, or in those with significant mitral regurgitation that is not suitable for repair, mitral valve replacement may be necessary. The threshold for Mitral Valve Surgery (commisurotmy or MVR) is higher than for PMBV in patients with Mitral Stenosis, and commisurotmy or repair is preferable to MVR, if feasible. Surgery for

moderate to severe Mitral Stenosis is indicated for symptomatic patients (New York heart association [NYHA] functional class iii or iv) where PMBV is unavailable or contraindicated (class i). MVR may also be considered for patients with severe MS and severe Pulmonary Hypertension with NYHA functional classes i or ii symptoms who are not candidates for PMBV or Mitral Valve Repair (class iia). Both mechanical and biologic prostheses are used for MVR; the choice of valve often depends on factors such as age, need for Concomitant Anticoagulation, and Left Ventricular (LV) size. Morbidity and mortality are higher with prosthetic valve replacement than with surgical or balloon valvotomy**.**

Definition and Causes

Mitral Regurgitation (MR) is leakage of blood from the left ventricle into the left atrium during systole. It is caused by various mechanisms related to structural or functional abnormalities of the mitral apparatus, adjacent myocardium, or both. The most common causes of mitral regurgitation are rheumatic heart disease, myxomatous degeneration, chordal rupture, infective endocarditis, coronary artery disease, and cardiomyopathy .

Pathophysiology and Natural History

Significant MR leads to volume overload of the left ventricle, because it has to accommodate both the stroke volume and regurgitant volume with each heartbeat. To compensate, the left ventricle dilates and becomes hyperdynamic. In acute severe MR, the left atrial and pulmonary venous pressures increase quickly, leading to pulmonary congestion and Pulmonary Edema. In chronic MR, a gradual increase in left atrial size and compliance compensate so that left atrial and pulmonary venous pressures do not increase until late in the course of the disease. Progressive left ventricular dilation eventually leads to an increase in afterload, contractile dysfunction, and heart failure. Left atrial enlargement predisposes the patient to Atrial Fibrillation and Arterial

Thromboembolism. In long-standing MR, patients may develop Pulmonary Hypertension and right-sided heart failure.

Signs and Symptoms

Patients with chronic, severe mitral regurgitation may remain asymptomatic for years because the regurgitant volume load is well tolerated as a result of compensatory ventricular and atrial dilation. When symptoms do develop, the most common are dyspnea, fatigue, orthopnea, paroxysmal nocturnal dyspnea, and palpitations caused by Atrial Fibrillation. Acute severe MR, as occurs with chordal rupture or papillary muscle rupture, is almost always symptomatic because the sudden regurgitant volume load in the nondilated left ventricle and atrium leads to Pulmonary venous hypertension and congestion.

The characteristic finding in a patient with MR is a blowing holosystolic murmur heard best at the cardiac apex. When ventricular enlargement is present, the apical impulse may be diffuse and laterally displaced, and a third heart sound may be heard.

Diagnosis

The chest radiograph demonstrates left atrial enlargement and cardiomegaly. Two-dimensional and Doppler echocardiography is indicated for all patients with suspected mitral regurgitation to confirm its presence and determine its severity (Class I). Two-dimensional echocardiography usually reveals the cause (e.g., the presence of myxomatous mitral valve disease and leaflet prolapse or evidence of underlying dilated cardiomyopathy). Evaluation of the severity of Mitral Regurgitation on echocardiography requires an integrated assessment of several parameters, including regurgitant jet size by Color Doppler, Regurgitant Jet Density by continuous-wave (CW) Doppler, and Pulmonary vein and Mitral Valve inflow by pulse-wave (PW) Doppler. Newer applications of Doppler echocardiography allow quantitative measurement of mitral regurgitation, including the regurgitant volume and the

regurgitant orifice area (ROA)—that is, the area through which the valve leaks in systole. In asymptomatic patients with significant mitral regurgitation, serial echocardiography every 6 to 12 months to assess LV size and systolic function is important for optimal timing of surgery (Class I). Transesophageal echocardiography is indicated for patients who are not adequately imaged by transthoracic echocardiography and before surgery to assess feasibility for repair (Class I). Stress echocardiography may be useful to assess exercise tolerance and the response of mitral regurgitation severity, Pulmonary pressure, and contractile reserve to exercise in asymptomatic patients with significant MR (Class IIa).

Cardiac catheterization is no longer routinely performed to evaluate mitral regurgitation severity, but it is indicated for those patients in whom noninvasive test results are inconclusive, and also to detect concomitant Coronary Artery Disease (CAD) in patients undergoing mitral valve surgery (Class I).

Treatment

Medical Treatment

In patients with acute severe MR, afterload reduction with intravenous nitroprusside and nitroglycerin reduces the regurgitant fraction and Pulmonary pressures. Placement of an intra-aortic balloon pump also helps stabilize these patients. However, these are temporary measures before urgent mitral valve repair or replacement. In patients with chronic asymptomatic Mitral Regurgitation caused by primary valve disease, there is no evidence for the routine use of medication in delaying the need for surgery or preventing left ventricular dysfunction. The management of these patients is focused on deciding on the appropriate timing of surgery, before the development of irreversible left Ventricular Dysfunction. Patients should be followed up every 6 to 12 months to assess for symptoms and to measure left ventricular size, function, and severity of MR by echocardiography (Class I).

In patients with ischemic heart disease or dilated cardiomyopathy, mitral regurgitation indicates a poor prognosis. MR in these patients is called functional mitral regurgitation and is caused by global or regional changes in left ventricular geometry as well as annular dilation. Functional MR is primarily treated medically with antihypertensive therapy, Angiotensin Converting Enzyme (ACE) inhibitors, beta blockers, diuretics, and antianginal therapies when mitral regurgitation is worsened by acute ischemia. Biventricular Pacing has also been shown to decrease the degree of mitral regurgitation in dilated cardiomyopathy.

Routine antibiotic prophylaxis for endocarditis is no longer recommended for patients with mitral regurgitation.

Surgery

Surgery is indicated for (1) symptomatic patients with severe primary MR (Class I) and (2) asymptomatic patients with severe primary MR and evidence of LV dysfunction (Class I). Optimal timing of mitral valve surgery is challenging in asymptomatic patients because the actual contractile function of the left ventricle is difficult to measure. The standard indications for surgery in asymptomatic patients are an LV end-systolic dimension of more than 4.0 cm and a resting LV ejection fraction of less than 60% (Class I). Other indications in asymptomatic patients include Pulmonary Hypertension or development of Atrial Fibrillation (Class IIa). In addition, mitral valve repair may be undertaken in experienced surgical centers for asymptomatic patients with severe MR, but without evidence of LV dilation or dysfunction, for which the likelihood of a successful repair is greater than 90% (Class IIa). Most asymptomatic patients with severe MR develop symptoms, LV dysfunction, or both over long-term follow-up. One retrospective study showed an increased risk of cardiac death (4%/year) in patients with severe mitral regurgitation based on an ROA of more than 0.4 cm^2. However, another recent prospective study has shown that careful follow-up of patients with severe MR and timing of surgery based on symptoms, LV dysfunction, development of Atrial Fibrillation, or Pulmonary Hypertension is associated with an excellent patient outcome.

In patients with severe functional mitral regurgitation, surgery may be considered for severe symptoms despite medical therapy. Patients with ischemic MR may improve with coronary bypass surgery if significant ischemia or myocardial viability is present. In many coronary bypass patients with MR, concomitant mitral valve repair with an undersized annuloplasty ring is performed. Patients with severe left ventricular dysfunction and significant MR were once believed to be poor surgical candidates, but recent studies have shown an acceptable operative risk. Symptoms usually improve, although a survival benefit has not been demonstrated.

The two available surgical options are mitral valve repair and mitral valve replacement. Mitral valve repair is the procedure of choice in the surgical management of MR caused by degenerative valve disease and in some cases of MR caused by infective endocarditis and ischemic heart disease. Repair offers several advantages over replacement, including lower operative time and long-term mortality, better preservation of LV function, a lower risk of subsequent infective endocarditis, and no need for long-term anticoagulation. Reoperation rates for mitral valve repair and replacement are similar, occurring at a rate of 1% to 2% per year. On the other hand, repair is technically more difficult than replacement, and many cases of mitral regurgitation are not amenable to valve repair. Percutaneous mitral valve repair is currently being investigated. The techniques involved include a clip that joins the mitral leaflets at their midpoint and an annuloplasty ring delivered via the coronary sinus.

Aims & Objectives

AIMS AND OBJECTIVES

- A Prospective study to analyze preoperative and postoperative Pulmonary Hypertension following mitral valvular heart disease using preoperative echocardiogram, intraoperative pressure studies and postoperative echocardiogram.

- To analyze the reduction in Pulmonary Hypertension following mitral valve replacement during patient follow up post surgically.

- To study the effect of pharmacological agents in reduction of Pulmonary Hypertension preoperatively and postoperatively in mitral valvular heart disease.

Materials & Methods

MATERIALS AND METHODS

A total of 265 patients were taken in this prospective study who came to the hospital with NYHA Class II-IV symptoms and with Mitral valvular heart lesions comprising of Mitral stenosis and Mitral Regurgitation with Pulmonary Hypertension from period of July 2007 –July 2009.

A detailed clinical examination and investigations were done on these patients and were categorized according to the study. All patients were prepared and consented before surgery as per the protocol and were operated under same conditions. All of them underwent Mitral valve replacement using St Jude's medical valve using cardiopulmonary bypass support.

A detailed clinical findings and preoperative values of these 265 patients are recorded over a structured proforma (Annexure) for all the patients.

All the patients are classified according to their age, sex, diagnosis, preoperative, intraoperative and post operative echo findings and result after surgery. The results thus obtained are tabulated and statistically analyzed and conclusions drawn thereafter.

Observations & Results

OBSERVATIONS AND RESULTS

265 Patients with mitral valvular heart disease and Pulmonary Hypertension who were admitted in government general hospital during July 2007-july 2010 were classified into different groups and tabulated according to different variables as follows:

AGE GROUP Vs PULMONARY HT SEVERITY PRE OPERATIVE

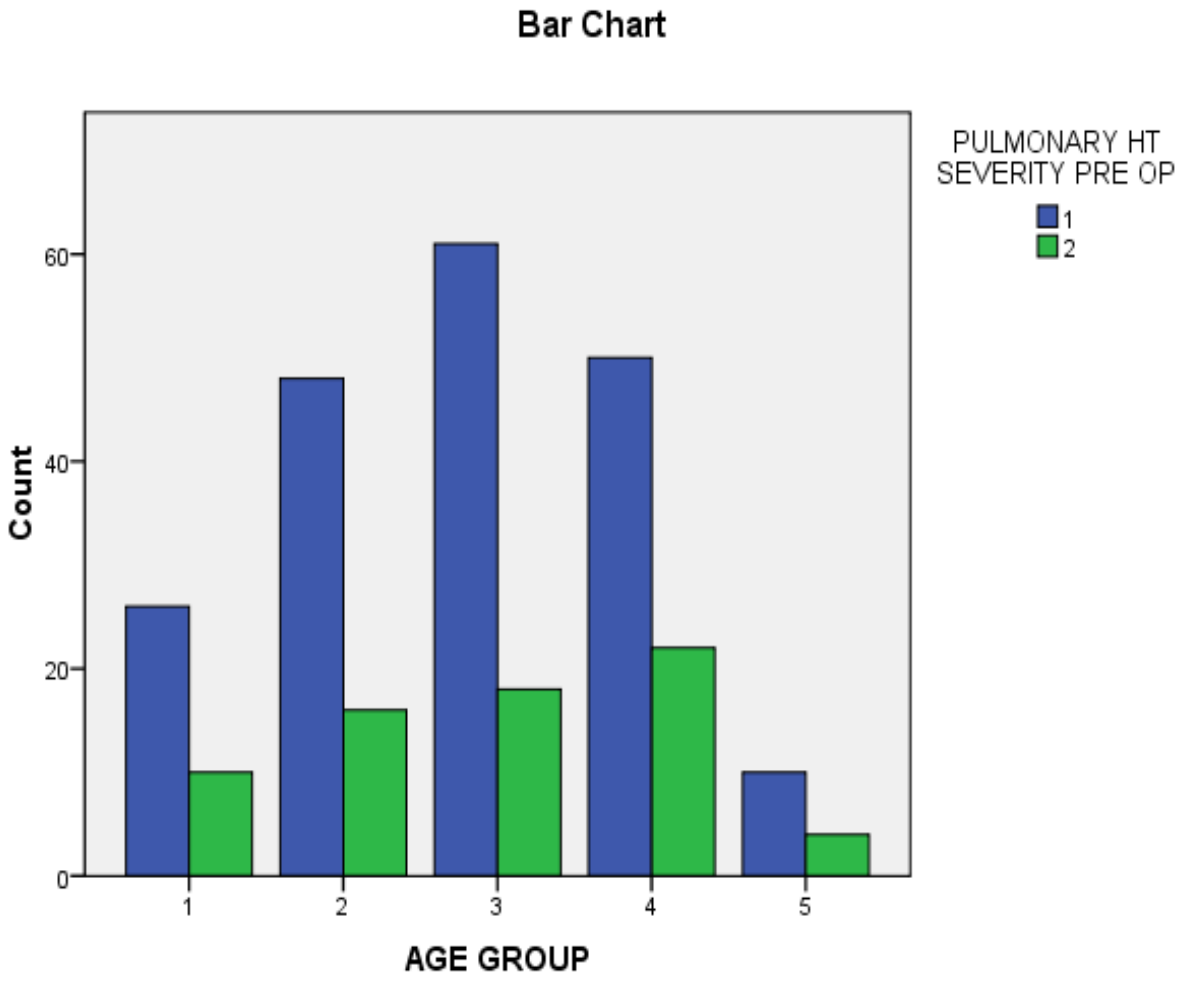
			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
AGE GROUP	1 (10-19)	Count	26	10	36
		% within PULMONARY HT SEVERITY PRE OP	13.3%	14.3%	13.6%
		% of Total	9.8%	3.8%	13.6%
	2 (20-29)	Count	48	16	64
		% within PULMONARY HT SEVERITY PRE OP	24.6%	22.9%	24.2%
		% of Total	18.1%	6.0%	24.2%
	3 (30-39)	Count	61	18	79
		% within PULMONARY HT SEVERITY PRE OP	31.3%	25.7%	29.8%
		% of Total	23.0%	6.8%	29.8%

Cont..

			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
	4 (40-49)	Count	50	22	72
		% within PULMONARY HT SEVERITY PRE OP	25.6%	31.4%	27.2%
		% of Total	18.9%	8.3%	27.2%
	5 (>50)	Count	10	4	14
		% within PULMONARY HT SEVERITY PRE OP	5.1%	5.7%	5.3%
		% of Total	3.8%	1.5%	5.3%
	Total	Count	195	70	265
		% within PULMONARY HT SEVERITY PRE OP	100.0%	100.0%	100.0%

Out of 265 patients analyzed 195 (73.58%) had severe Pulmonary Hypertension and remaining 70 patients (26.42 %) had moderate to mild Hypertension. Out of 265 patients, age group which had affected maximum with PHT was found to be in the 3rd to 4th decade which is about 31.3% which is a significant productive group. Next group to be affected by this severe PHT was found to be in the 4th to 5th decade (18.9 %).Age group which had affected the least was found in the 5th decade and beyond.

Chi-Square Tests			
	Value	df	Asymmetrical. Sig. (2-sided)
Pearson Chi-Square	1.304[a]	4	.861
On performing the chi-square test there was no significant correlation between the age group and severe Pulmonary Hypertension.			



1) 10-19 Yrs
2) 20-29 Yrs
3) 30-39 Yrs
4) 40-49 Yrs
5) >50 Yrs

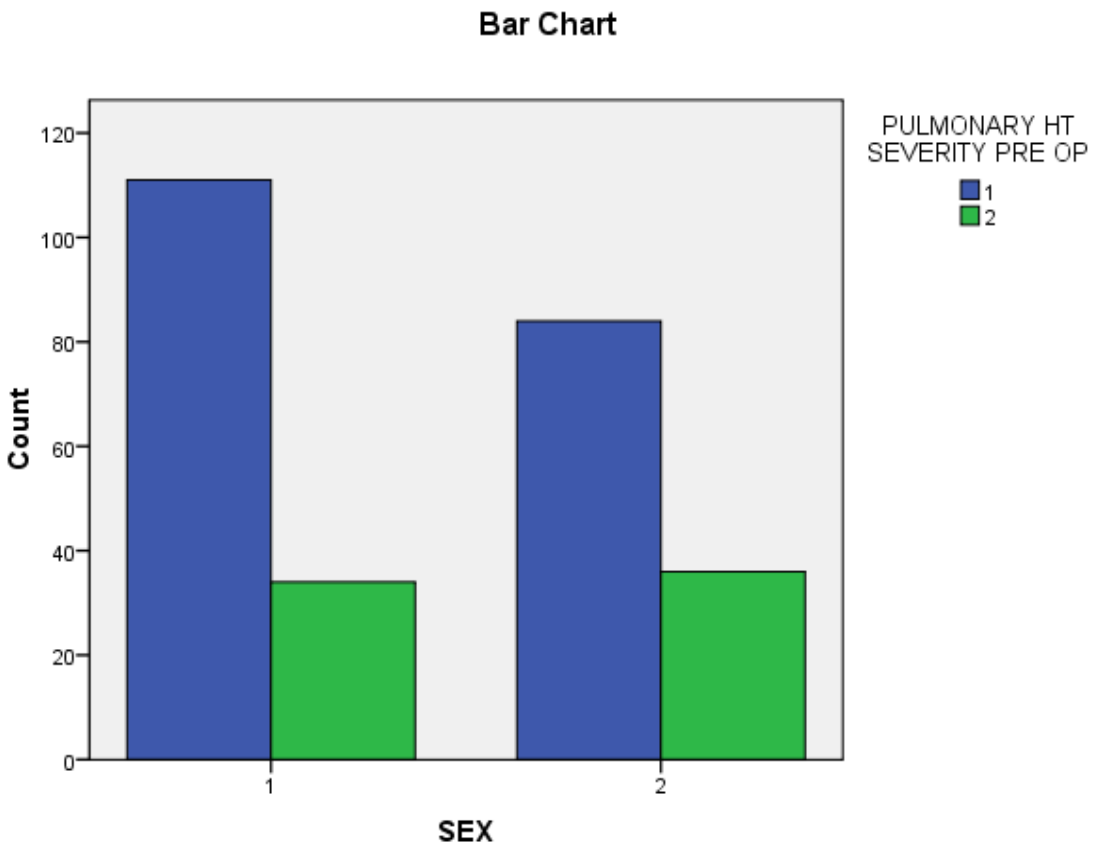
SEX DISTRIBUTION

SEX Vs PULMONARY HT SEVERITY PRE OPERATIVE					
			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
SEX	1 (Female)	Count	111	34	145
		% within PULMONARY HT SEVERITY PRE OP	56.9%	48.6%	54.7%
		% of Total	41.9%	12.8%	54.7%
	2 (Male)	Count	84	36	120
		% within PULMONARY HT SEVERITY PRE OP	43.1%	51.4%	45.3%
		% of Total	31.7%	13.6%	45.3%
Total		Count	195	70	265
		% within PULMONARY HT SEVERITY PRE OP	100.0%	100.0%	100.0%
		% of Total	73.6%	26.4%	100.0%

According to this study the female patients were affected more with severe Pulmonary Hypertension which constitutes about (41.9%) and male patients constituted about 31.7% out of the 195 patients who had severe Pulmonary Hypertension. On an average about 73.6% of patients had severe Pulmonary Hypertension taking both males and females into consideration. The distribution for mild to moderate disease was found to be equal.

Statistical Method	Value	df	Asymmetric Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.450[a]	1	.229		

On performing the chi-square test there was no significant statistical correlation between sex and Pulmonary Hypertension.



1) MALE 2) FEMALE

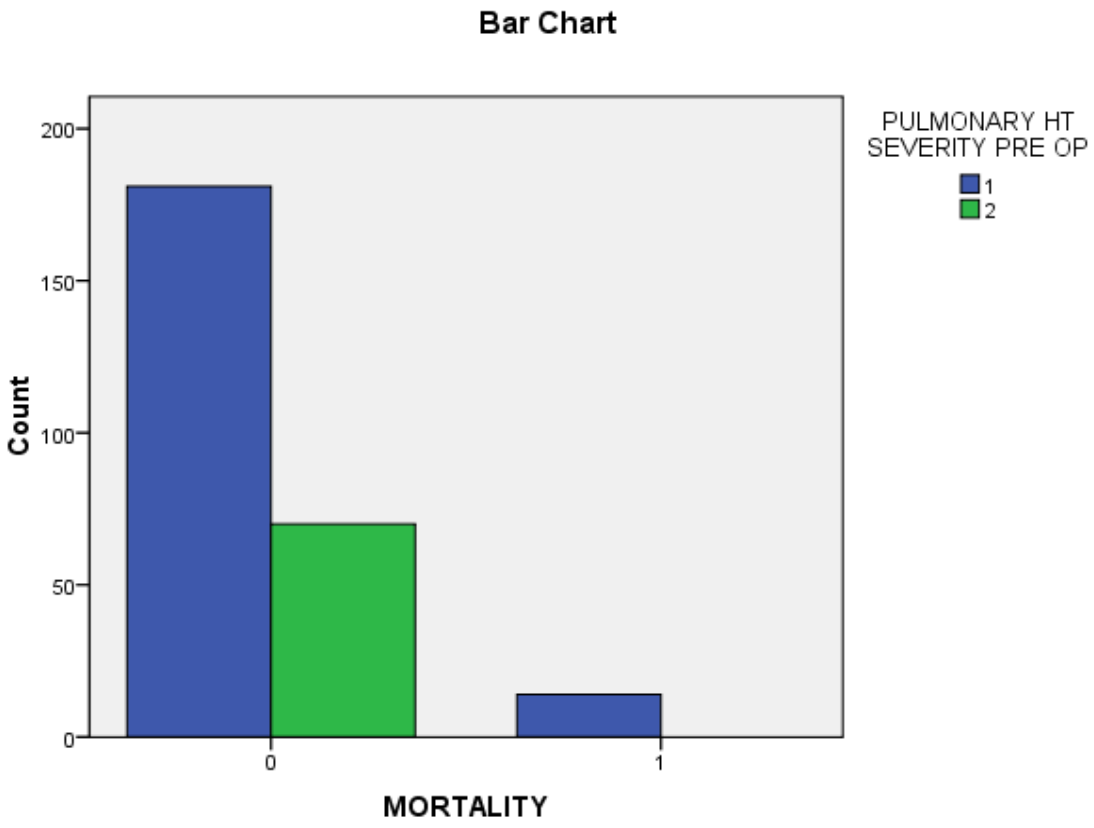
MORTALITY AND PULMONARY HYPERTENSION

PULMONARY HT SEVERITY PRE OPERATIVE			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
MORTALITY	0	Count	181	70	251
		% within PULMONARY HT SEVERITY PRE OP	92.8%	100.0%	94.7%
		% of Total	68.3%	26.4%	94.7%
	1	Count	14	0	14
		% within PULMONARY HT SEVERITY PRE OP	7.2%	.0%	5.3%
		% of Total	5.3%	.0%	5.3%
	Total	Count	195	70	265
		% within PULMONARY HT SEVERITY PRE OP	100.0%	100.0%	100.0%
		% of Total	73.6%	26.4%	100.0%

On analyzing mortality in relation to Pulmonary Hypertension there were 14 deaths (5.3%) out of total number of 265 patients and which includes about 7.2% of 195 patients known to have severe Pulmonary Hypertension. About 94.7% patients had good postoperative outcome following this surgery done for severe PHT associated with Rheumatic Mitral valve disease.

Statistical Method	Value	df	Asymmetric. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	5.306[a]	1	.021		

On performing the chi-square test there was a significant correlation between severe Pulmonary Hypertension and mortality which is a well known factor and this study also confirms the hypothesis.



1- No Mortality

2-Mortality

PATIENTS IN NYHA POST MVR (NYHA Vs PULMONARY HT SEVERITY PRE OPERATIVE)

PULMONARY HT SEVERITY PRE OPERATIVE			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
NYHA	1	Count	6	48	54
		% within PULMONARY HT SEVERITY PRE OP	3.3%	68.6%	21.5%
		% of Total	2.4%	19.1%	21.5%
	2	Count	172	22	194
		% within PULMONARY HT SEVERITY PRE OP	95.0%	31.4%	77.3%
		% of Total	68.5%	8.8%	77.3%
	3	Count	3	0	3
		% within PULMONARY HT SEVERITY PRE OP	1.7%	.0%	1.2%
		% of Total	1.2%	.0%	1.2%
	Total	Count	181	70	251
		% within PULMONARY HT SEVERITY PRE OP	100.0%	100.0%	100.0%

PULMONARY HT SEVERITY PRE OPERATIVE			Severity Levels 1- Severe 2-Non-Severe		
			1	2	Total
NYHA	1	Count	6	48	54
		% within PULMONARY HT SEVERITY PRE OP	3.3%	68.6%	21.5%
		% of Total	2.4%	19.1%	21.5%
	2	Count	172	22	194
		% within PULMONARY HT SEVERITY PRE OP	95.0%	31.4%	77.3%
		% of Total	68.5%	8.8%	77.3%
	3	Count	3	0	3
		% within PULMONARY HT SEVERITY PRE OP	1.7%	.0%	1.2%
		% of Total	1.2%	.0%	1.2%
	Total	Count	181	70	251
		% within PULMONARY HT SEVERITY PRE OP	100.0%	100.0%	100.0%
		% of Total	72.1%	27.9%	100.0%

About 77.3% of patients were in NYHA classification II following this surgery and about 21.5% were in NYHA class I followed by 1.2% in NYHA Class III.

REDUCTION OF PULMONARY HYPERTENSION

T-Test

Group Statistics

	SEX	N	Mean	Std. Deviation	Std. Error Mean
PRE-OPERATIVE PULMONARY HT	1	145	72.15	19.993	1.660
	2	120	69.34	20.756	1.895
0-MONTHS PULMONRY HT	1	145	45.50	11.775	.978
	2	120	46.77	10.744	.981
3-MONTHS PULMONARY HT	1	134	37.56	6.245	.539
	2	117	39.58	8.237	.762
6-MONTHS PULMONARY HT	1	134	29.78	5.356	.463
	2	117	30.97	6.515	.602

On analyzing the data the Pulmonary Hypertension was reduced to a mean of approximately 46.14mmHG immediately following surgery and significant reduction of about 30.38 mmHG after 6 months of surgery. At 3 months follow up the average reduction in PHT was about 38.57mmHG .This analysis showed that the reduction in PHT had a gradual course after an initial reduction from a mean PHT value of 70.75 mmHG preoperative values. This gives us a clue that the reduction in Pulmonary Hypertension is a gradual one and needed change in organic level as well as in the dynamic level which takes some time to achieve.

Independent Samples Test

		t-test for Equality of Means		
		df	Sig.(2-tailed)	Mean Difference
PRE OP PULMONARY HT	Equal variances assumed	263	.264	2.810
	Equal variances not assumed	250.067	.266	2.810
0-MONTHS PULMONRY HT	Equal variances assumed	263	.364	-1.270
	Equal variances not assumed	260.472	.360	-1.270
3-MONTHS PULMONARY HT	Equal variances assumed	249	.028	-2.021
	Equal variances not assumed	214.527	.031	-2.021
6-MONTHS PULMONARY HT	Equal variances assumed	249	.111	-1.198
	Equal variances not assumed	224.980	.116	-1.198

On performing the T-test on the given statistics there was a statistical significance in patients who had a reduction in Pulmonary Hypertension after 3 months postoperatively from the preoperative value.

Preoperative Hemodynamic in Patients with Pulmonary Hypertension

Variable	Severe PHT
Systolic PAP (mm Hg) [range]	84.5 [75–105]
Mean PAP (mm Hg) [range]	70.88 [51–90]

An average systolic pressure of 84.5mmHg and a mean PAP of 70.88 mmHg were found in this study.

INTRAOPERATIVE PHT

S.NO	MALE	FEMALE
PAP (MEAN)	62.8mmHg	73.6 mmHg

An average mean PAP of 62.8mmHg and 73.6mmHg were observed in male and female patients respectively with severe Pulmonary Hypertension intraoperatively.

POST MVR PHT (6 MONTHS)

S.NO	MALE	FEMALE
MEAN PAP	30.97mmHg	29.78mmHg

An average 30.97 mmg and 29.78mmHg were recorded as mean PAP in male and female patients respectively following Mitral valve replacement after an observation of 6 months.

PAP AT DAY 0

S.NO	MALE	FEMALE
MEAN PAP	46.77mmHg	45.5mmHg

At day zero the average reduction in PAP were found to be about 46.77mmHg and 45.5mmHg in both male and female patients respectively following MVR. This is found to be significant observation as the immediate drastic reduction in PHT as we expect following valve replacement does not occur as anticipated and gives us obvious clues as to the reasons behind the reduction in Pulmonary arterial pressures.

IMMEDIATE POSTOPERATIVE DEATH

S.NO	MALE	FEMALE
DEATH	04	10

An average of 7.07 %% of death in this study showed death in the immediate postoperative period which includes day 0 and day 1in patients with severe Pulmonary Hypertension .

MEAN ACC TIME AND CPB TIME

S.NO	ACC	CPB
LEFT ATRIAL APPROACH	56.6MINS	84 .56 MINS
SEPTAL APPROACH	68.2 MINS	100.68MINS

This table shows the average Aortic Cross Clamp time and Cardiopulmonary Bypass Time needed for the procedure. It is evident from the table that the septal approach takes a longer ACC and CPB time on comparison.

In all the 265 patients who underwent MVR, 195 of them had severe Pulmonary Hypertension. All of them had the classical left atrial approach through Sondergaard's groove incision except 11 cases which was approached through the septum after opening the right atrium.

All cases were opened through the standard median sternotomy and were done using cardiopulmonary bypass utilizing cardioplegic arrest to open the chamber (s).

In about 195 out of 265 patients (73.58%), MVR was done with either partial are complete chordal preservation. All the patients had St Jude's valve placed as the mechanical prosthetic valve. Except for the 14 deaths which occurred due to low cardiac output failures all other cases were weaned of the ventilator by day 1 and from the inotropic supports by day 2 or day 3.

Those patients who had severe Pulmonary Hypertension who underwent MVR were followed up using echo at day 0 ,3 months and 6 months later and results thus obtained showed an early reduction in about 21.16% of males and 27.35% of females .About 79.84% of male patients and 72.65% of female patients showed a trend of reduction in Pulmonary Hypertension in about 3- 6 months time as compared to those patients who showed an immediate reduction in Pulmonary Hypertension following mitral valve replacement for mitral valvular heart disease with severe Pulmonary Hypertension .

Discussions

DISCUSSION

Pulmonary Hypertension (PH or PHT) is an increase in blood pressure in the Pulmonary artery, Pulmonary vein, or Pulmonary capillaries, together known as the lung vasculature, leading to shortness of breath, dizziness, fainting, and other symptoms, all of which are exacerbated by exertion. Pulmonary Hypertension can be a severe disease with a markedly decreased exercise tolerance and heart failure. It was first identified by Dr. Ernst von Romberg in 1891. According to the most recent classification, it can be one of five different types: *arterial, venous, hypoxic, thromboembolic* or *miscellaneous*

SIGNS AND SYMPTOMS

Because symptoms may develop very gradually, patients may delay seeing a physician for years. Common symptoms are shortness of breath, fatigue, non-productive cough, angina pectoris, fainting or syncope, peripheral edema (swelling around the ankles and feet), and rarely hemoptysis (coughing up blood).

Pulmonary venous hypertension typically presents with shortness of breath while lying flat or sleeping (orthopnea or paroxysmal nocturnal dyspnea), while Pulmonary arterial hypertension (PAH) typically does not.

DIAGNOSIS

A physical examination is performed to look for typical signs of Pulmonary Hypertension, including a loud P2 (pulmonic valve closure sound), (Para) sternal heave, jugular venous distension, pedal edema, ascites, hepatojugular reflux, clubbing etc. Evidence of tricuspid insufficiency is also sought and, if present, is consistent with the presence of Pulmonary Hypertension.

Investigations include apart from the blood routine, an ECG, Echocardiogram to confirm diagnosis and quantify the severity, x-ray chest which shows the classical picture of dilated and prominent Pulmonary arteries with obliterated left cardiac shadow with straightening of left cardiac border. Arterial blood gas analysis or simple

bedside 02 saturation may give us the clue for the diagnosis. Biopsy of the lung is usually not indicated unless the Pulmonary Hypertension is thought to be due to an underlying interstitial lung disease. But lung biopsies are fraught with risks of bleeding due to the high intrapulmonary blood pressure. Blood BNP level is also being used now to follow progress of patients with Pulmonary Hypertension.

Cardiac catheterization is not routinely done nowadays to quantify Pulmonary Hypertension as newer Echocardiography studies throw us sufficient lights for the evidence and measurement of Pulmonary Hypertension.

Normal Pulmonary arterial pressure in a person living at sea level has a mean value of 12–16 mm Hg (1600–2100 Pa). Pulmonary Hypertension is present when mean Pulmonary artery pressure exceeds 25 mm Hg (3300 Pa) at rest or 30 mm Hg (4000 Pa) with exercise.

Mean Pulmonary artery pressure (mPAP) should not be confused with systolic Pulmonary artery pressure (sPAP), which is often reported on echocardiogram reports. A systolic pressure of 40 mm Hg typically implies a *mean* pressure more than 25 mm Hg. Roughly, mPAP = 0.61•sPAP + 2.

Vascular resistance is a term used to define the resistance to flow that must be overcome to push blood through the circulatory system. The resistance offered by the peripheral circulation is known as the systemic vascular resistance (SVR), while the resistance offered by the vasculature of the lungs is known as the Pulmonary vascular resistance (PVR).

Units for measuring vascular resistance are dynes·s·cm-5 or Pascal seconds per cubic meter (Pa.s/m^3). Pediatric cardiologists use hybrid reference units (HRU), also known as Wood units, as they were introduced by Dr. Paul Wood. To convert from dynes·s·cm-5 to Wood units you must divide by 80.

Normal Values For Vascular Resistance		
Systemic vascular resistance	1170 ± 270 dynes·s·cm^{-5}	117 ± 27 MPa.s/m^3
Systemic vascular resistance index	2130 ± 450 dynes·s·cm^{-5}·m^2	213 ± 45 MPa.s/m
Pulmonary vascular resistance	67 ± 30 dynes·s·cm^{-5}	6.7 ± 3 MPa.s/m^3

1 Calculation of resistance

The basic tenet of calculating resistance is that flow is equal to driving pressure divided by resistance.

CAUSES AND CLASSIFICATION

A 1973 meeting organized by the World Health Organization was the first to attempt classification of Pulmonary Hypertension. A distinction was made between primary and secondary PH, and primary PH was divided in the "arterial plexiform", "veno-occlusive" and "thromboembolic" forms. A second conference in 1998 at Évian-les-Bains also addressed the causes of secondary PH (i.e. those due to other medical conditions), and in 2003, the 3rd World Symposium on Pulmonary Arterial Hypertension was convened in Venice to modify the classification based on new understandings of disease mechanisms. The revised system developed by this group provides the current framework for understanding Pulmonary Hypertension. The system includes several improvements over the former 1998 Evian Classification system. Risk factor descriptions were updated, and the classification of congenital systemic-to Pulmonary shunts was revised. A new classification of genetic factors in PH was recommended, but not implemented because available data were judged to be inadequate.

The Venice 2003 Revised Classification system can be summarized as follows:

WHO Group I - Pulmonary arterial hypertension (PAH)

Idiopathic (IPAH)

Familial (FPAH)

Associated with other diseases (APAH): collagen vascular disease (e.g. scleroderma), congenital shunts between the systemic and Pulmonary circulation, portal hypertension, HIV infection, drugs, toxins, or other diseases or disorders

Associated with venous or capillary disease

WHO Group II - Pulmonary Hypertension associated with left heart disease

Atrial or ventricular disease

Valvular disease (e.g. mitral stenosis)

WHO Group III - Pulmonary Hypertension associated with lung diseases and/or hypoxemia

Chronic obstructive Pulmonary Disease (COPD), Interstitial Lung Disease (ILD)

Sleep-disordered breathing, alveolar hypoventilation

Chronic exposure to high altitude

Developmental lung abnormalities

WHO Group IV - Pulmonary Hypertension due to chronic thrombotic and/or embolic disease

Pulmonary Embolism in the proximal or distal Pulmonary Arteries

Embolization of other matter, such as tumor cells or parasites

WHO Group V - Miscellaneous

PATHOGENESIS

Whatever the initial cause**,** Pulmonary arterial hypertension (WHO Group I) involves the vasoconstriction or tightening of blood vessels connected to and within the lungs. This makes it harder for the heart to pump blood through the lungs, much as it is harder to make water flow through a narrow pipe as opposed to a wide one. Over time, the affected blood vessels become both stiffer and thicker, in a process known as

fibrosis. This further increases the blood pressure within the lungs and impairs their blood flow. In addition, the increased workload of the heart causes thickening and enlargement of the right ventricle, making the heart less able to pump blood through the lungs, causing right heart failure. As the blood flowing through the lungs decreases, the left side of the heart receives less blood. This blood may also carry less oxygen than normal. Therefore it becomes harder and harder for the left side of the heart to pump to supply sufficient oxygen to the rest of the body, especially during physical activity.

Pathogenesis in Pulmonary venous hypertension (WHO Group II) is completely different. There is no obstruction to blood flow in the lungs. Instead, the left heart fails to pumps blood efficiently, leading to pooling of blood in the lungs. This causes Pulmonary edema and pleural effusions.

In hypoxic Pulmonary Hypertension (WHO Group III), the low levels of oxygen are thought to cause vasoconstriction or tightening of Pulmonary arteries. This leads to a similar pathophysiology as Pulmonary arterial hypertension.

In chronic thromboembolic Pulmonary Hypertension (WHO Group IV), the blood vessels are blocked or narrowed with blood clots. Again, this leads to a similar pathophysiology as Pulmonary arterial hypertension.

TREATMENT

Treatment is determined by whether the PH is arterial, venous, hypoxic, thromboembolic, or miscellaneous. Since Pulmonary venous hypertension is synonymous with congestive heart failure, the treatment is to optimize left ventricular function by the use of diuretics, beta blockers, ACE inhibitors, etc., or to repair/replace the mitral valve or aortic valve.

In PAH, lifestyle changes, digoxin, diuretics, oral anticoagulants, and oxygen therapy are considered conventional therapy, but have never been proven to be beneficial in a randomized, prospective manner.

A number of agents has recently been introduced for primary and secondary PAH. The trials supporting the use of these agents have been relatively small, and the only measure consistently used to compare their effectivity is the "6 minute walking test". Many have no data on mortality benefit or time to progression.

VASOACTIVE SUBSTANCES

Many pathways are involved in the abnormal proliferation and contraction of the smooth muscle cells of the Pulmonary arteries in patients with Pulmonary arterial hypertension. Three of these pathways are important since they have been targeted with drugs — endothelin receptor antagonists, phosphodiesterase type 5 inhibitors[15], and prostacyclin derivatives.

Because inexpensive generic drugs for this disease are not widely available, the World Health Organization does not include them in its model list of essential medicines.

PROSTAGLANDINS

Prostacyclin (prostaglandin I_2) is commonly considered the most effective treatment for PAH. Epoprostenol (synthetic prostacyclin, marketed as Flolan) is given via continuous infusion that requires a semi-permanent central venous catheter. This delivery system can cause sepsis and thrombosis. Flolan is unstable, and therefore has to be kept on ice during administration. Since it has a half-life of 3 to 5 minutes, the infusion has to be continuous (24/7), and interruption can be fatal. Other prostanoids have therefore been developed. Treprostinil (Remodulin) can be given intravenously or subcutaneously, but the subcutaneous form can be very painful. An increased risk of sepsis with intravenous Remodulin has been reported by the CDC. Iloprost (Ilomedin) is also used in Europe intravenously and has a longer half life. Iloprost (marketed as Ventavis) is the only inhaled form of prostacyclin approved for use in the US and Europe. This form of administration has the advantage of selective deposition in the lungs with less systemic side effects. Oral and inhaled forms of

Remodulin are under development. Beraprost is an oral prostanoid available in Japan and South Korea. ENDOTHELIN RECEPTOR ANTAGONISTS

The dual (ET_A and ET_B) endothelin receptor antagonist bosentan (marketed as Tracleer) was approved in 2001. Sitaxentan, a selective endothelin receptor antagonist that blocks only the action of ET_A, has been approved for use in Canada, Australia, and the European Union, to be marketed under the name Thelin. Sitaxentan has not been approved for marketing by the U.S. Food and Drug Administration (FDA). A new trial to address the FDA's concerns had begun in 2008. A similar drug, ambrisentan is marketed as Letairis in U.S. In addition, another dual/nonselective endothelin antagonist, Actelion-1, from the makers of Tracleer, had entered clinical trials in 2008.

PHOSPHODIESTERASE TYPE 5 INHIBITORS

Sildenafil, a selective inhibitor of cGMP specific phosphodiesterase type 5 (PDE5), was approved for the treatment of PAH in 2005. It is marketed for PAH as Revatio. We had used this medication in about 73 (195) of the patients with severe Pulmonary Hypertension in our study.

ACTIVATORS OF SOLUBLE GUANYLATE CYCLASE

Soluble guanylate cyclase (sGC) is the intracellular receptor for NO. As of April 2009, the sGC activators cinaciguat and riociguat are undergoing clinical trials for the treatment of PAH

Treatment for hypoxic and miscellaneous varieties of Pulmonary Hypertension has not been established. However, studies of several agents are currently enrolling patients. Many physicians will treat these diseases with the same medications as for PAH, until better options become available. Such treatment is called off-label.

MONITORING

Patients are normally monitored through commonly available tests such as:

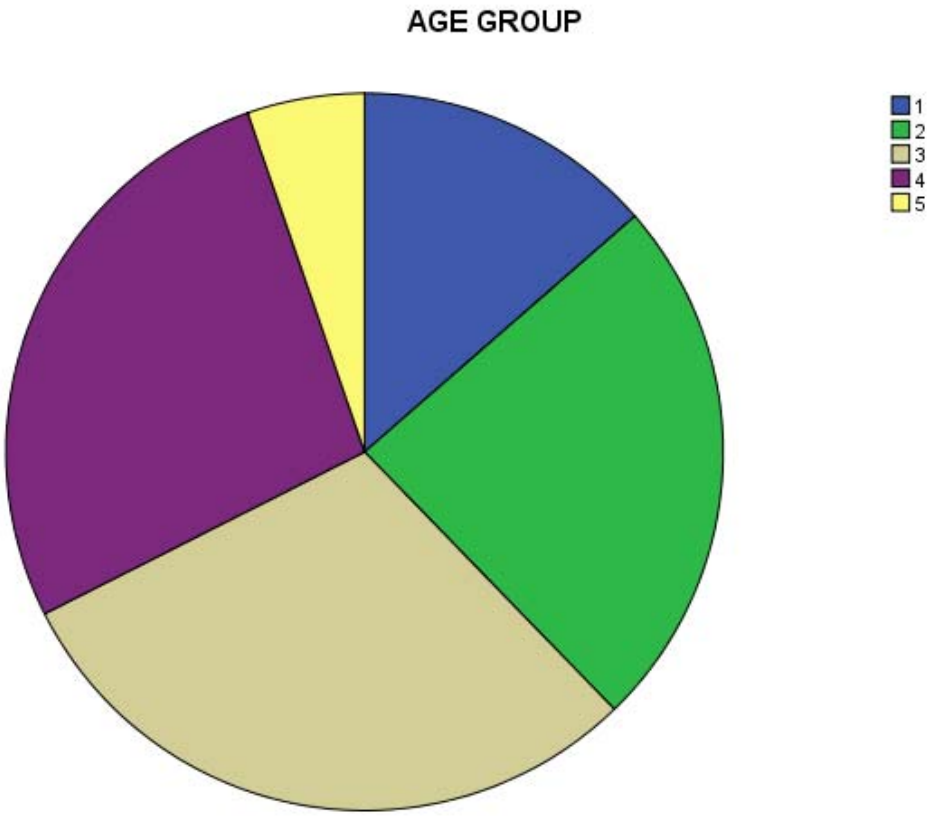
- Pulse oximetry,
- Arterial blood gas tests,
- Chest X-rays,
- Serial ECG tests,
- Serial echocardiography.

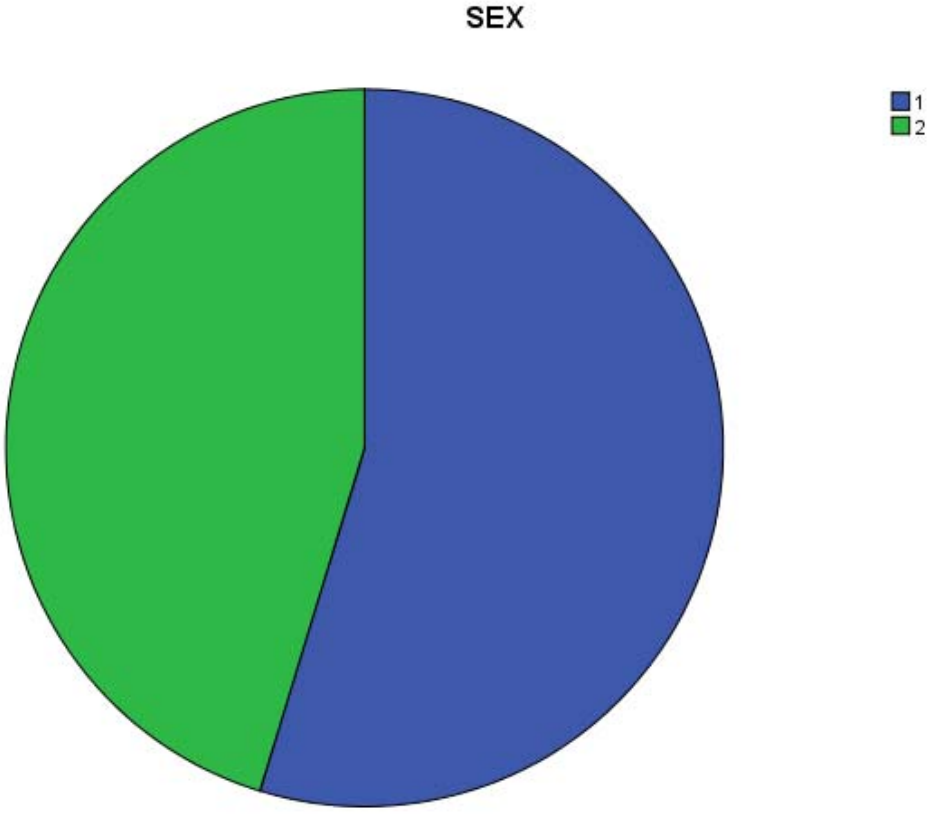
In all our 265 patients who underwent MVR 195 had severe Pulmonary Hypertension and females 111/195(56.9%) outnumbered the males in this diagnosis. The immediate reduction of Pulmonary Hypertension happened in only approximately 21 % of the patients and the remaining had persistent PHT which showed a fall progressively following a 6 months period suggesting the various factors which might be responsible for the Pulmonary Hypertension.

The average mortality noted in this study was comparatively less (7.2%) % than an expected value of about 10-15% by the previous studies. The difference might be because of the earlier decision to operate upon this severe group before irreversible factors sets in and also due to the better intraoperative and postoperative care using latest agents so far approved for this purpose.

However the long-term follow up of these patients are needed to conclude firmly regarding the use of newer agents used to reduce Pulmonary Hypertension either intraoperatively are postoperatively.

17 patients among the total patients required a septal approach due to the concomitant presence of tricuspid regurgitation and hence the need for devega procedure done for them.


AGE GROUP
1
2
3
4
5


SEX
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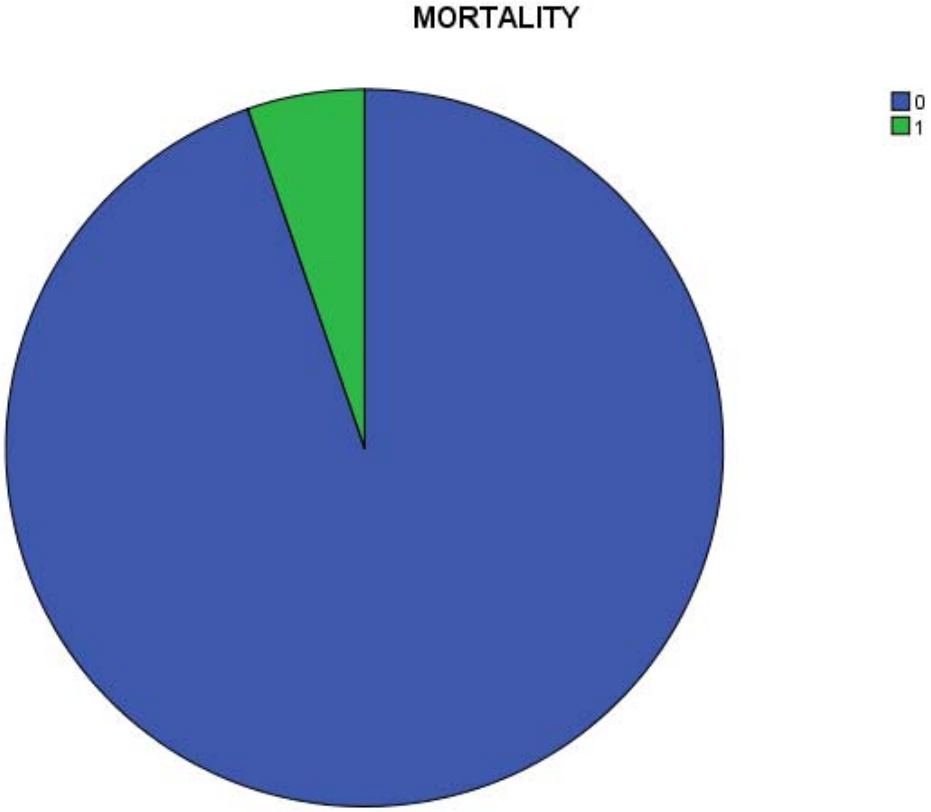
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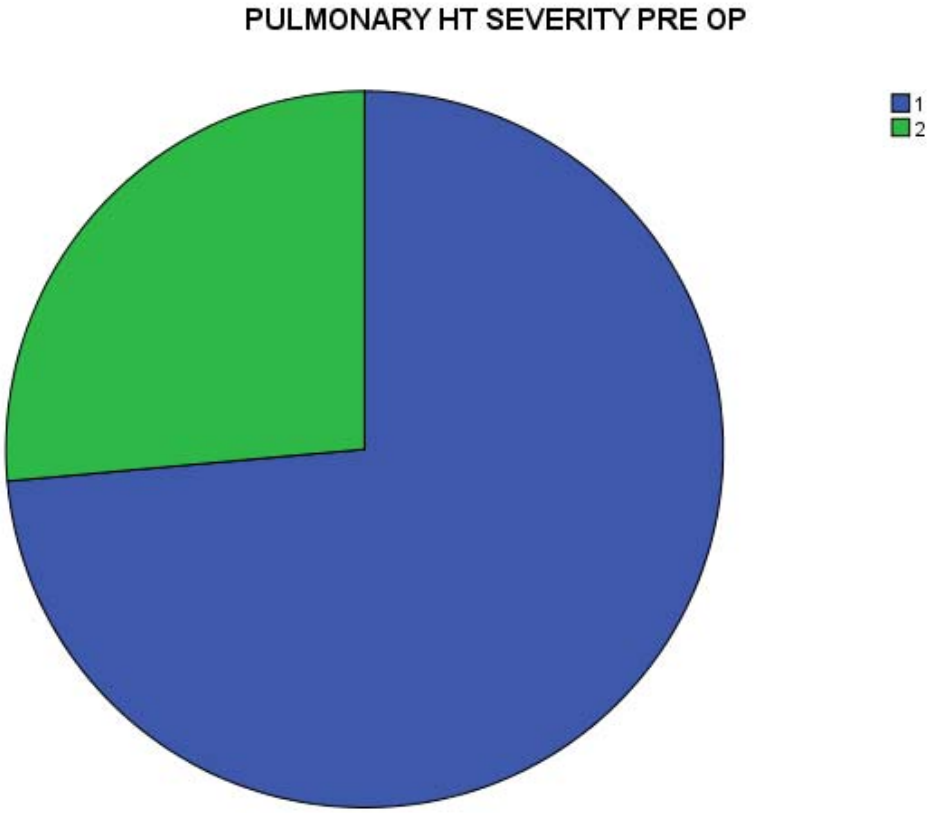
PULMONARY HT SEVERITY PRE OP
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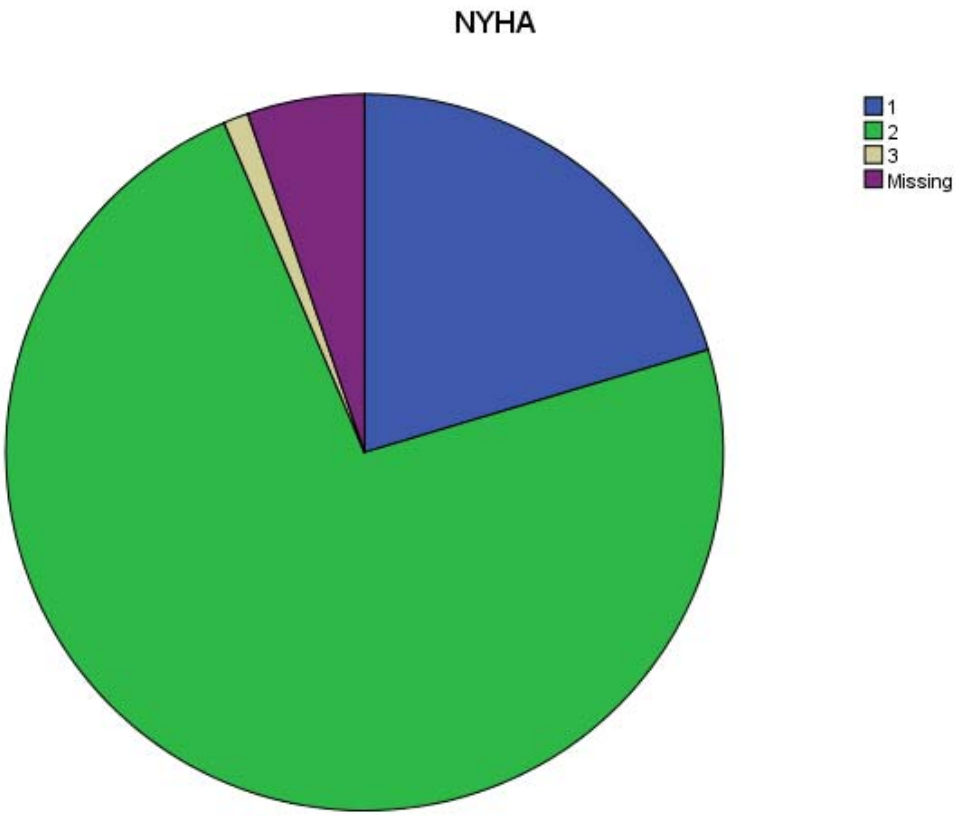
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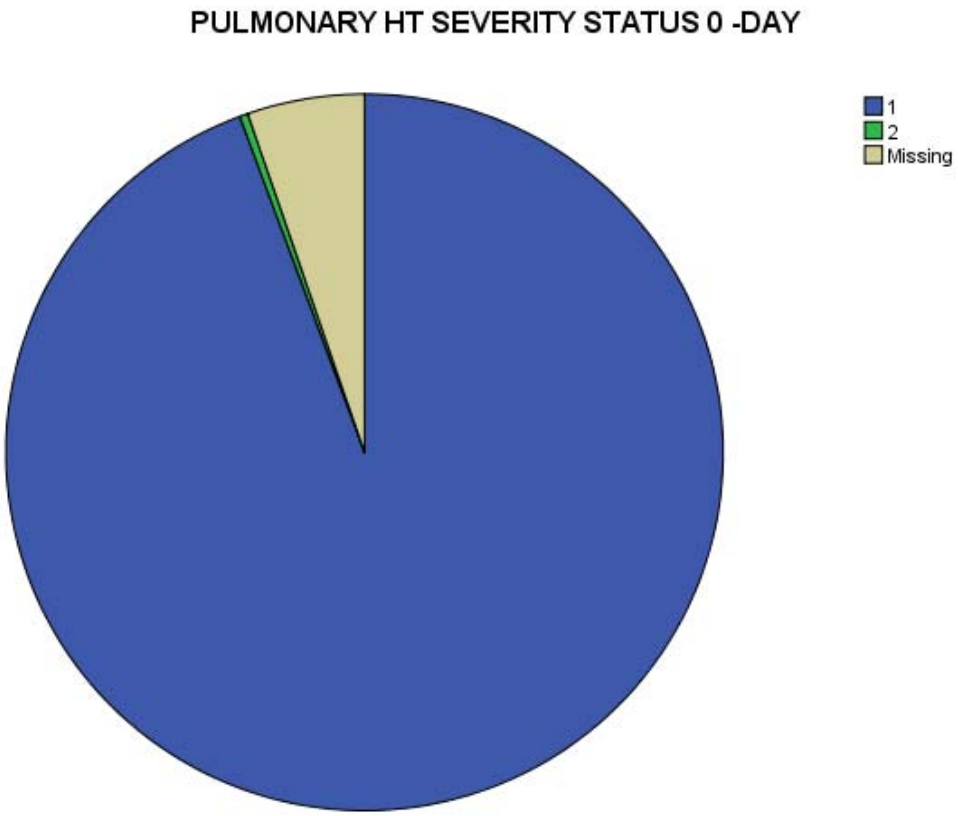
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2
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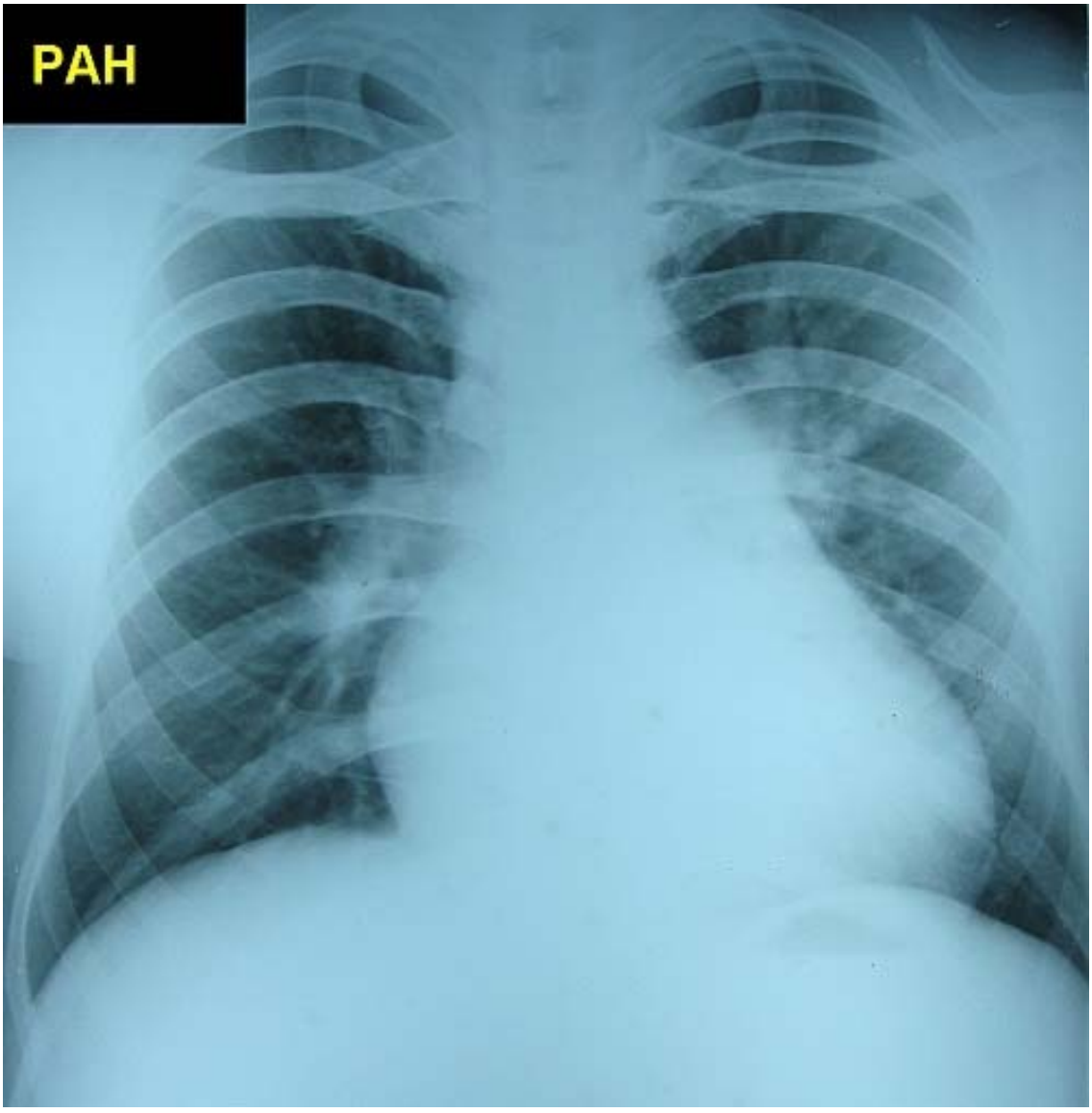
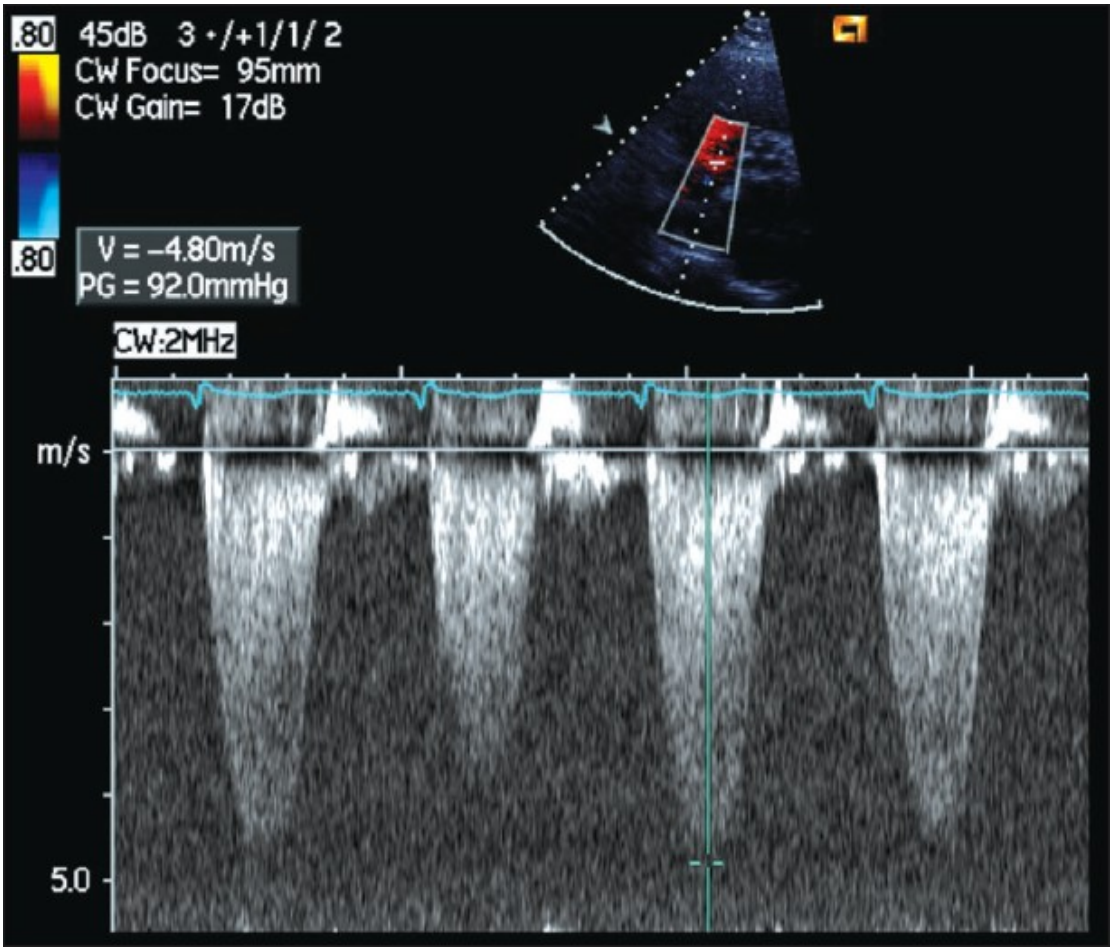


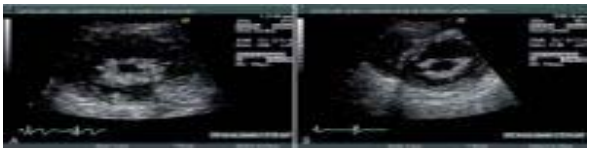
CHEST X-RAY OF SEVERE PULMONARY HYPERTENSION

C

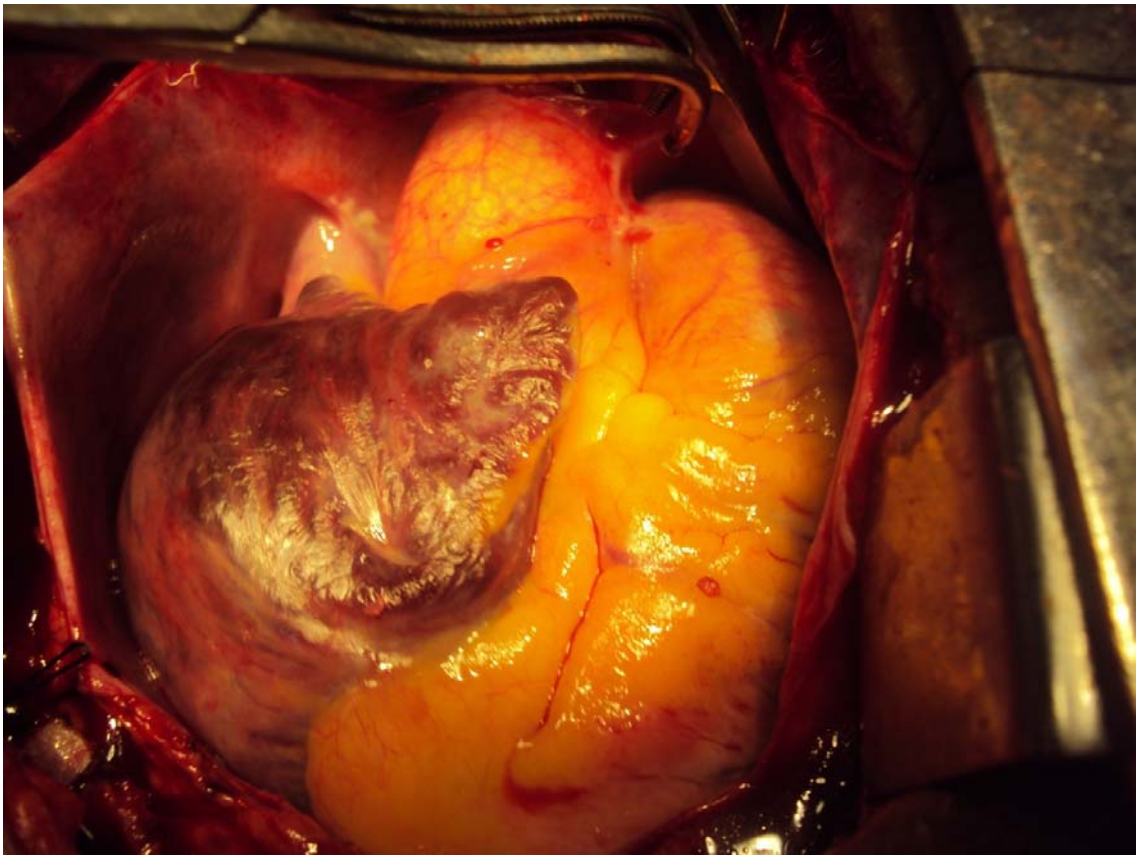


ECHO DOPPLER PICTURE OF SEVERE PULMONARY HYPERTENSION

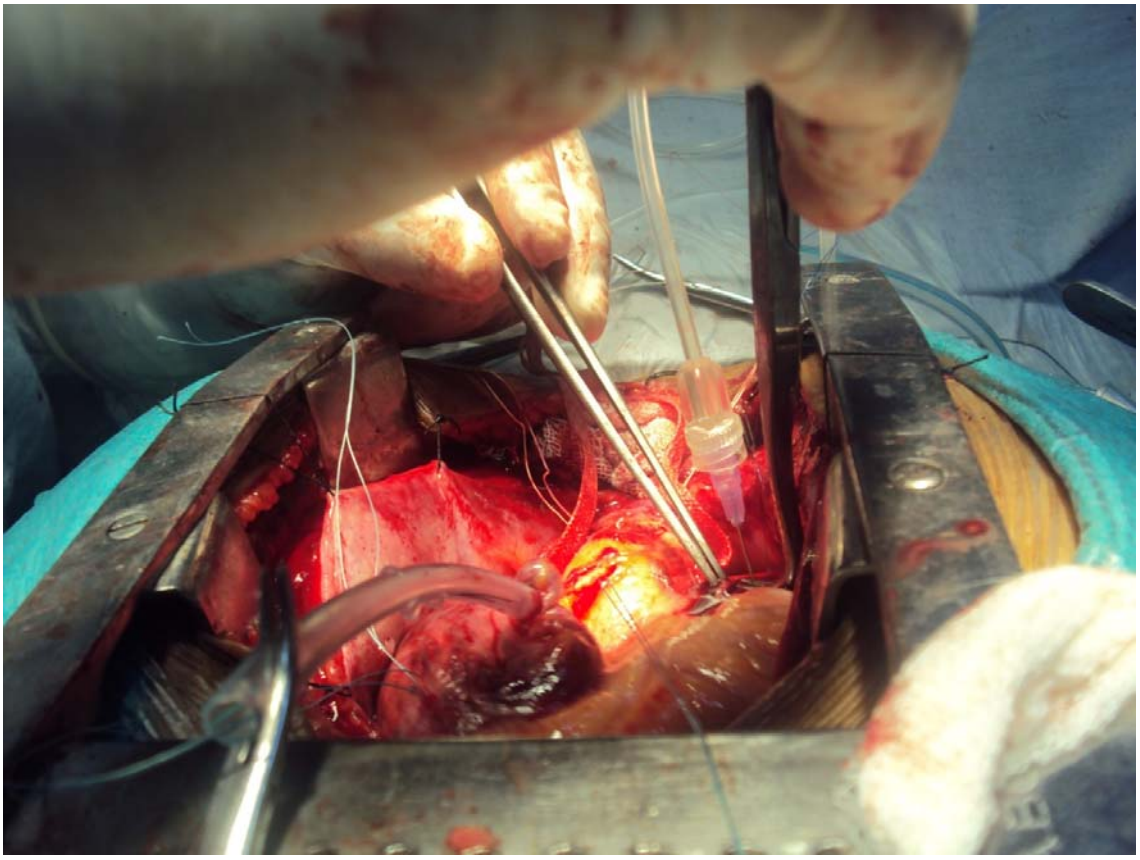
EXCISED SPECIMEN OF MITRAL VALVE



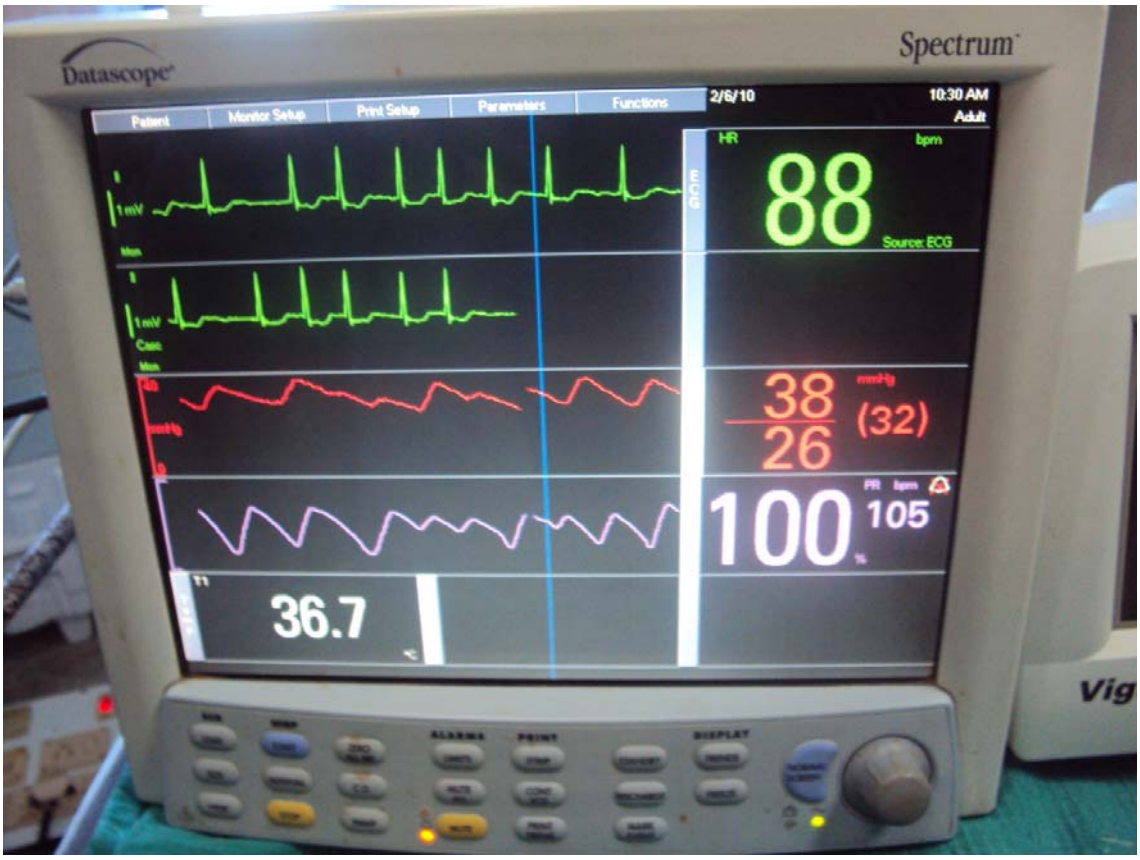
DOPPLER IMAGE OF RHEUMATIC MITRAL STENOSIS



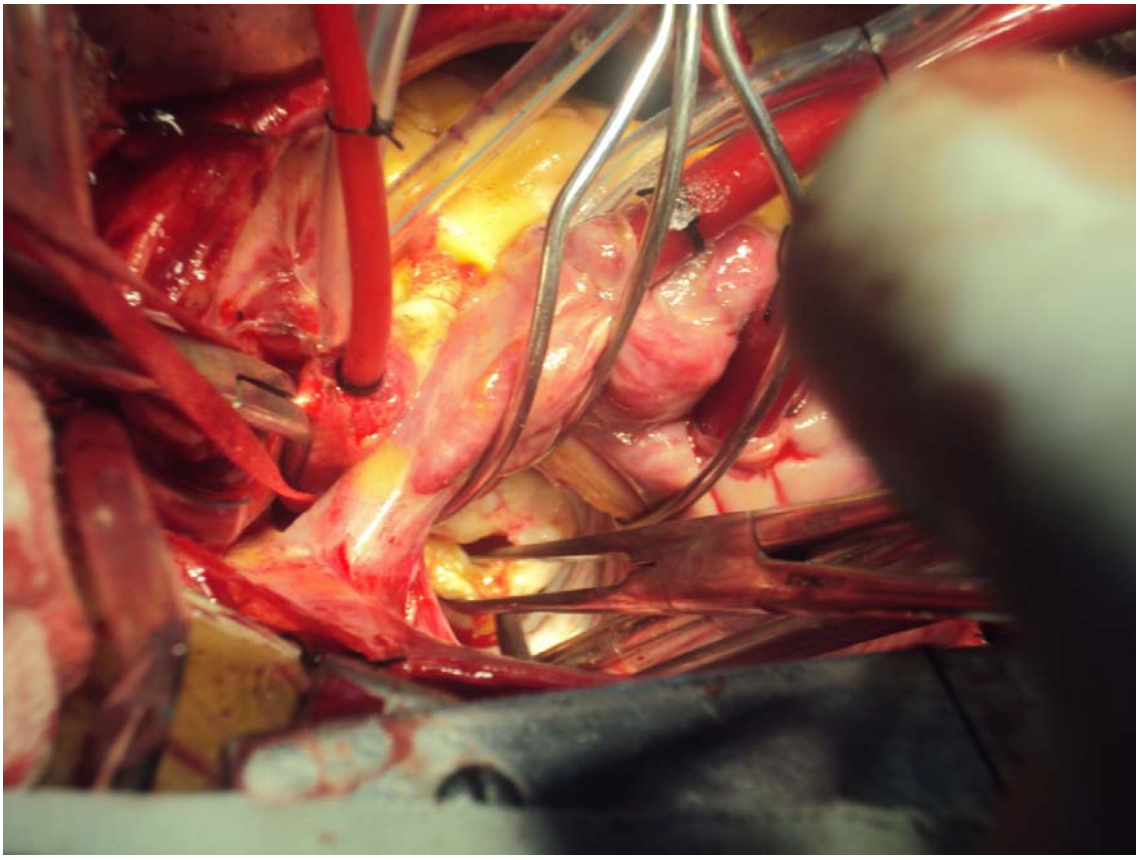
PRE-CARDIOPULMONARY BYPASS PICTURESHOWING DILATED MAIN PULMONARY ARTERY



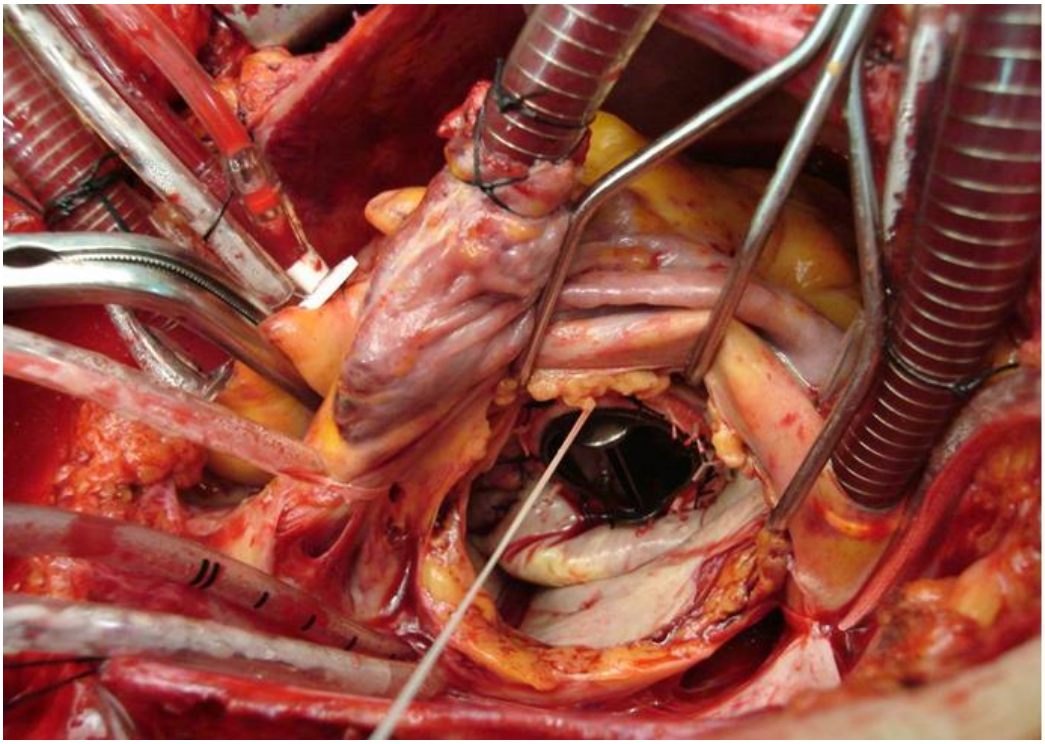
INTRAOPERATIVE PULMONARY PRESSURE MEASUREMENT USING NEEDLE



INTRAOPERATIVE PULMONARY PRESSURE RECORDING



PHOTOGRAPH SHOWING RHEUMATIC MITRAL VALVE EXPOSURE ON CARDIOPULMONARY BYPASS



INTRAOPERATIVE PICTURE SHOWING INSERTION OF St JUDE'S MECHANICAL PROSTHETIC VALVE IN MITRAL POSITION

Summary & Conclusion

SUMMARY &CONCLUSION

Pulmonary arterial hypertension has long been considered a risk factor for poor outcome in patients undergoing MVR, with operative mortality ranging from 15%–31%. Najafi and colleagues found the degree of PAH correlated strongly with perioperative mortality, ranging from 16% in patients with mild PAH to 23% in severe PAH and 61% when PAP was at systemic levels. Recently, several reports have demonstrated improved outcome in patients with PAH undergoing MVR, with perioperative mortality ranging from 2.3%–10%.The improved outcome was attributed to better myocardial preservation, preservation of the subvalvular apparatus, and improved postoperative care. In our study, the overall operative mortality rate was 7.07 % which is consistent with recent reports. However, the mortality rate in patients with PHT overall was 5.5 % which is better than the recent reports.

Numerous studies have examined hemodynamic changes in this subset of patients at different intervals after mitral valve procedures. Most have demonstrated an immediate reduction in PAP and PVR, signifying a sudden drop in left atrial pressure and reversal of the severe spastic Pulmonary vasoconstriction that accompanies left atrial hypertension in some patients. Others have shown slow regression of elevated PAP and PVR several months postoperatively. These reports point toward the involvement of multiple factors in the development of PAH in mitral valve disease. There have been studies of closed mitral commissurotomy and balloon mitral valvotomy in this subgroup of patients from India, which have shown good results in terms of survival, postoperative functional class, and hemodynamics. In our study the mean PAP and PVR did not fall significantly immediately following MVR .The mean fall in PAP was about 46.77 % in males and 45.5% in females which was against the previous views .Although the mean PAP fell significantly from 84.5 to 70.88% mm Hg, it remained within the definition of severe PAH. The PVR showed no significant reduction immediately after MVR, but a gradual regression was seen over a 6 months period and the fall was significant at 6 months period when compared with the preoperative values. This indicates the reactive component of Pulmonary arterial

vasoconstriction, which may be responsible for part of the disproportionate elevation of PVR seen in as many as 20% of patients undergoing mitral valve procedures.

The persistence of residual elevated PAP and PVR well beyond the normal limit in patients suggests an irreversible component of the increased PVR. Other variables that determine the immediate and long-term results of surgery in this subset of patients include advanced age, acute presentation, decreased left ventricular ejection fraction, functional class, right heart failure and increased left ventricular end-diastolic pressure. Vincens and colleagues identified clinical right heart failure as a predictor of operative mortality, and both right ventricular systolic pressure and right ventricular hypertrophy as predictors of poor outcome. Others have identified severe tricuspid regurgitation and the need for concomitant tricuspid surgery as risk factors for operative mortality in this population. In this study, 47% of patients had right ventricular hypertrophy and/or dilatation, and 33% had severe tricuspid regurgitation.

Despite the high operative mortality in most series of MVR in patients with severe PAH, a striking improvement in survival was noted. In our series, functional class improved by one class or more in the majority of survivors. Long-term morbidity was related mainly to anticoagulation and was attributed to poor patient compliance due to illiteracy in this part of the world. Repair of the mitral valve in patients with predominant MR could have avoided these complications but it was not undertaken because of the high rate of repair failure in patients with rheumatic etiology of mitral regurgitation as well as severe subvalvular pathology with calcification of leaflets in most of them.

I acknowledge that the lack of follow-up of Pulmonary vascular dynamics by catheterization constitutes a limitation of this study and was related primarily to economic factors. A postoperative lung biopsy might have added to the information, but this was not undertaken as most of the patients refused consent for it.

I conclude that MVR is safe and effective even in the presence of severe PAH as long as the Pulmonary arterial pressures are below systemic pressures. With supra-systemic PAP, MVR carries a high risk of mortality, and the patient continues to have

persistent PAH in the postoperative period. Significant reduction in PHT following MVR takes place only gradually and in this study about 6 months period showed a decline in PHT as compared to the previous belief of immediate reduction in PHT in majority of the patients.

This explains the multifactorial causes of PHT in patients with Rheumatic mitral valvular heart disease and severe Pulmonary Hypertension.

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Proforma

PROFORMA

NAME: **AGE:** **SEX:**

OCCUPATION: **DATE OF ADMISSION:**

ADDRESS FOR COMMUNICATION:

ADMISSION DIAGNOSIS: MS MS+MR MR LA CLOT

BRIEF HISTORY : DOE ANGINA PALPITATION PND ORTHOPNOEA OLIGURIA PE

NYHA CLASS : 1 2 3 4 5

RHEUMATIC HEART DISEASE : YES NO

H/0 ANTIFAILURE MEDICATION: YES NO

GENERAL EXAMINATION : HT WT HR BP
A /ICTERUS/CYANOSIS/PEDAL OEDEMA/JVP

CVS EXAMINATION : RV IMPULSE LV IMPULSE AF a) present b) absent.

HEART SOUNDS : S1 S2 S1 LOUD VARIABLE SOFT

S3 S4 YES NO

P2 LOUD, SOFT

CARDIAC MURMERS : MDM GRADE

PSM GRADE

RESPIRATORY SYSTEM : BAE ; ADDED SOUNDS
a) RHONCHI b) FINE CREPTS

ABDOMEN : HEPATOMEGALY

SPLENOMEGALY

ASCITES

CNS : NEUROLOGICAL DEFICIT.

INVESTIGATIONS : BLOOD TC DC ESR PCV Hb PL COUNT

BT CT

BLOOD UREA BLOOD SUGAR S. CREATININE S. Na k

ECG RVH P mitrale LVH AF ST-T SEGMENT OTHERS

X-RAY CHEST :

CT RATIO :

BLOOD GROUPING & Rh Typing

ECHO: MVO MVA LEAFLET MOBILITY THICKENING Ca + SCF

LA SIZE **PHT** **a) mild** **b)** **moderate** **c) severe**

LV DIMENSION **EF:**

CORONARY ANGIOGRAM:

INTRA OP DETAILS: TOTAL CPB TIME **TOTAL 'X ' CLAMP TIME**

CP 1 2 3 4

VALVE SIZE :

PULMONARY ARTERY PRESSURE SYSTOLE: **DIASTOLE:**

POST OP DETAILS: PULMONARY ARTERY PRESSURE SYSTOLE: DIASTOLE:

POSTOP : VENTILATOR SUPPORT : <24 HRS 24-48 HRS 48-72 HRS >72 HRS

INOTROPES : DOP/DOB DOP+DOB DOP/DOB+ADR/ISO OTHERS

COMPLICATIONS : SSSI STERNAL DEHISCENCE LCOS MODS

MORTALITY : LCOS ARRHYTHMIA SUDDEN CARDIAC ARREST

IMMEDIATE POSTOPERATIVE : PULMONARY ARTERY PRESSURE :

3 MONTHS

6 MONTHS

1 YEAR.

MASTER CHART

S.No	NAME	AGE	AGE GROUP*	DURATION SEX	SEX**	DEATH	PREOP PHT (mmHg)	0 MON (mmHg)	3 MON (mmHg)	6 MON (mmHg)	SEVERITY NYHA	NYHA***
1	GANDHIMATHY	45	4	1	1	0	80	40	35	25	1	2
2	MD.ALI	27	2	2	2	0	70	38	32	30	1	2
3	NASRUDEEN	47	4	1	2	0	75	48	30	26	1	2
4	VARALAXMI	42	4	2	1	0	86	52	40	36	1	2
5	ANNATHAI	35	3	2	1	0	78	50	44	30	1	1
6	RAJAMNI	27	2	1	2	0	74	46	40	36	1	2
7	RAJAMAL	29	2	2	1	0	38	30	26	20	1	1
8	KARTHIK	25	2	1	2	0	36	30	24	20	1	1
9	CHANDRA	40	4	2	1	0	88	48	38	30	1	2
10	NADIYA	18	1	2	1	0	78	44	40	36	1	2
11	BALA	30	3	3	2	0	90	54	40	36	1	2
12	MALATHI	20	1	1	1	0	40	32	26	24	1	1
13	RANI	50	5	2	1	0	86	46	40	30	1	2
14	RAMAR	24	2	2	2	0	80	50	40	32	1	2
15	LAKSHMI	45	4	2	1	0	74	46	44	30	1	2
16	PARTHIBAN	27	2	1	2	0	40	32	30	26	1	2
17	MARYRANI	35	3	1	1	0	36	30	28	20	1	2
18	MANJULA	35	3	2	1	0	76	44	40	32	1	2
19	LAKSMIDEVI	45	4	1	1	0	82	46	40	32	1	2
20	MANI	45	4	2	2	0	78	52	46	34	1	2
21	SEKAR	43	4	3	2	0	40	36	30	22	1	2
22	NAGARAJ	48	4	2	2	1	94	88			0	
23	RADHABAI	49	4	1	1	0	76	48	44	34	1	2

24	MANIKANDAN	16	1	3	2	0	40	36	30	28	1	2
25	BALASUBRAMANI 2	27	2	2	2	0	80	56	52	34	1	2
26	RENUKA	20	2	2	1	0	36	30	28	20	1	1
27	SANTHOSH	19	1	2	2	0	68	54	48	40	1	1
28	ELUMALAI	50	5	2	2	0	38	34	30	22	1	2
29	RAMACHANDRA	15	1	2	2	0	88	52	46	38	1	2
30	PONNAMAL	47	4	2	1	0	40	32	30	20	1	2
31	SIVAJIRAO 4	44	4	1	2	0	78	50	46	38	1	2
32	SAROJA 4	49	4	2	1	0	34	30	22	20	1	2
33	UMA	38	3	2	1	0	90	40	34	30	1	2
34	MEENAKSI	28	2	1	1	0	88	38	36	26	1	2
35	ANJALI	30	3	2	1	0	90	48	40	32	1	2
36	THIIRUMATHY	15	1	2	1	0	30	28	24	20	1	2
37	UMAPATHY	45	4	2	2	0	72	56	44	36	1	2
38	INDRANI	34	3	2	1	0	70	44	30	28	1	2
39	SARAVANAN 2	25	2	2	2	0	40	38	36	30	1	1
40	ANURADHA	32	3	2	1	0	84	44	36	30	1	2
41	MANJULA	43	4	3	1	0	90	48	40	36	1	2
42	KUMAUDHA	21	2	2	1	0	98	46	40	38	1	2
43	EZHIL 4	45	4	2	2	0	84	58	50	46	1	2
44	SANGEETHA 2	22	2	2	1	0	70	40	38	30	1	2
45	KALAIVANI 2	26	2	2	1	0	82	42	40	30	1	2
46	RAMASAMI	29	2	2	2	0	86	44	38	30	1	2
47	KUMAR 4	40	4	2	2	0	40	36	30	24	1	2
48	KAVERI	47	4	2	1	0	90	44	38	34	1	2
49	MURUGAN	32	3	1	2	0	70	44	32	30	1	2
50	SUBRAMANI	42	4	2	2	0	78	48	40	32	1	2
51	SHANTHI	45	4	2	1	0	88	40	32	28	1	2
52	MURUGAIAN	52	5	2	2	1	80	74				
53	GOMATHY	40	4	3	1	0	36	30	28	20	1	2
54	AMARAVATHY	45	4	2	1	0	42	36	30	22	1	1
55	PRABU	21	2	1	2	0	78	50	44	32	1	2
56	VEERAMANI	36	3	2	2	0	78	44	38	26	1	3
57	KUMARAN	35	3	2	2	0	42	40	36	20	1	2
58	ANNAKILI	26	2	2	1	0	74	46	42	36	1	2
59	MANI	30	3	1	2	0	80	54	50	38	1	2

60	NALINI	25	2	1	1	0	76	48	46	32	1	2
61	SUBAKUTI	30	3	2	2	0	40	36	30	22	1	1
62	MASTHAN	45	4	2	1	0	78	44	38	36	1	2
63	JEELAN	40	4	2	2	0	36	34	30	20	1	1
64	PETER	50	5	2	2	0	40	34	30	20	1	1
65	SUMATHY	26	2	1	1	0	78	40	34	24	1	2
66	SHANTHI	34	3	2	1	0	76	44	42	30	1	2
67	HARI	45	4	2	2	0	36	30	28	20	1	1
68	KOLA	15	1	2	1	0	72	40	38	26	1	2
69	MANGAI	35	3	1	1	0	42	36	30	22	1	1
70	SARASWATHY 4	45	4	1	1	0	50	48	40	38	1	2
71	DHANALAKSMI	53	5	2	1	0	30	28	26	20	1	1
72	MOHAN	39	3	2	2	0	94	56	50	42	1	2
73	SELVI	43	4	2	1	0	80	42	40	36	1	2
74	CHANDRASEKAR 4 1	47	4		2	0	88	54	50	34	1	2
75	NANDHINI	13	1	1	1	0	80	60	48	38	1	3
76	AMBA	30	3	2	1	0	72	44	40	32	1	2
77	SHANMUGAVALI 40 4	40	4		1	0	76	40	38	26	1	2
78	KALA 1	18	1	2	1	0	82	40	36	22	1	2
79	NADHIYA	18	1	2	1	0	70	46	40	32	1	2
80	DILLIBABU 4	48	4	2	2	0	42	32	30	24	1	1
81	VARADHARAJ 3	39	3	1	2	0	40	34	30	26	1	1
82	KUMAR	27	2	2	2	0	76	52	48	36	1	2
83	KALAVATHY	44	4	1	1	0	90	48	42	36	1	2
84	SILAMBARASAN	27	2	2	2	0	74	52	50	40	1	2
85	PERUMAYEE	47	4	3	1	0	40	36	30	26	1	1
86	ALGUMANI	42	4	2	2	0	90	48	40	36	1	2
87	SHANU	35	3	2	1	0	78	50	46	24	1	2
88	MUNIAMMAL	42	4	2	1	0	86	48	40	36	1	2
89	ATHIRUBAN 3	39	3	2	2	0	78	44	40	32	1	1
90	NATRAJAN 2	27	2	1	2	0	30	26	20	18	1	1
91	RAJAMMAL	45	4	2	1	0	74	40	36	30	1	1
92	MURUGAN	18	1	2	2	0	78	40	42	32	1	2
93	VENKATESAN	44	4	2	2	0	80	46	38	26	1	2
94	DURAISAMI	43	4	2	2	0	40	38	36	24	1	1
95	GOPI	16	1	1	2	0	80	46	40	38	1	2
96	ELANGOVAN	39	3	2	2	0	74	50	44	34	1	2
97	AYYAPAN	33	3	1	2	0	36	30	28	20	1	1
98	SALEEMA	22	2	1	1	0	40	32	30	24	1	2
99	ELANGOVAN	39	3	3	2	0	76	54	48	34	1	2
100	SAHINBEGUM	46	4	2	1	0	80	48	40	34	1	2
101	SHANTHI	21	2	2	1	0	72	40	36	30	1	2
102	SELVI	18	1	2	1	0	68	40	38	30	1	2
103	RAJI	33	3	2	1	0	80	48	40	32	1	2
104	DHATCHANAMOORTHI	25	2	1	2	0	78	48	44	36	1	2
105	PARTHASARATHI	35	3	2	2	0	36	30	28	22	1	2
106	SEKAR	44	4	2	2	0	80	48	40	38	1	2
107	SHAJIN	46	4	2	1	0	74	48	43	37	1	1
108	SEKAR	44	4	1	2	0	80	58	48	38	1	2

109	SARVANAN	47	4	2	2	0	76	42	38	30	1	2
110	RAJESVARI	14	1	2	1	0	40	35	28	22	1	2
111	DEIVANI	52	5	3	1	0	80	48	47	35	1	2
112	IYYAPAN	20	2	2	2	0	40	30	28	22	1	1
113	ESAKKIAPPAN	40	4	1	2	0	70	48	40	36	1	2
114	PONNAN	45	4	2	2	0	80	58	50	36	1	3
115	LAKSMIDEVI	18	1	2	1	0	70	40	38	33	1	2
116	SUGANTHI	35	3	2	1	0	76	44	39	27	1	2
117	NADIYA	26	2	1	1	0	30	26	20	18	1	1
118	MADAN	15	1	4	2	0	34	30	27	23	1	1
119	ARUN	23	2	2	2	0	40	36	30	22	1	1
120	LALITHA	29	2	2	1	0	78	48	40	42	1	2
121	RANI	28	2	1	1	0	88	40	38	30	1	2
122	SHANMUGAM	42	4	2	2	0	36	32	30	25	1	1
123	JANAKIRAMAN	15	1	1	2	0	86	58	50	32	1	2
124	MAHESVARI	24	2	2	1	0	88	46	40	32	1	2
125	VATCHALA	59	5	2	1	0	86	44	42	38	1	2
126	NANDHINI	30	3	2	1	0	38	34	30	24	1	1
127	GOVINDAN	33	3	2	2	0	88	48	40	37	1	2
128	MARINESAN	53	5	1	2	0	88	49	47	35	1	2
129	VASANTHI	30	3	2	1	0	40	38	30	28	1	2
130	PERIASAMI	49	4	2	2	0	38	32	30	25	1	1
131	KOLANCHIAPPAN	38	3	1	2	0	90	54	47	33	1	2
132	VIDHYA	17	1	2	1	0	44	39	27	20	1	1
133	VASANTHI	25	2	1	1	0	32	30	27	22	1	1
134	DILIP	23	2	2	2	0	80	49	44	38	1	2
135	RAMADURAI	36	3	2	2	0	88	49	42	31	1	2
136	THANGAKODI	61	2	1	1	0	40	38	34	31	1	1
137	GEETHA	31	3	2	1	0	80	48	40	36	1	2
138	AYYASAMI	26	2	2	2	0	80	59	47	34	1	2
139	KESAVAN	37	3	2	2	0	88	53	45	33	1	2
140	MURUGESAN	36	3	1	2	0	90	56	50	37	1	2
141	THENMOZHI	38	3	2	1	0	92	46	44	33	1	2

142	MURUGAMMAL	20	2	2	1	0	90	56	49	37	1	2
143	BASKAR	18	1	2	2	0	88	49	43	37	1	2
144	MINNALKODI	35	3	2	1	0	90	48	42	36	1	2
145	PANDURANGAN	36	3	2	2	0	40	36	30	24	1	1
146	SIVAPRAKASH	40	4	2	2	0	32	28	20	18	1	1
147	DEVI	23	2	1	1	0	86	46	40	38	1	2
148	SIVAGAMI	40	4	2	1	0	40	32	30	24	1	1
149	YASODHA	54	5	2	1	0	86	48	40	34	1	2
150	CHINNARAJ	15	1	2	2	0	46	36	30	22	1	1
151	SARASWATHY 4	35	3	1	1	0	94	48	40	32	1	2
152	RAJA	18	1	2	2	0	90	50	42	36	1	2
153	SHANTHI	23	2	1	1	0	86	40	36	30	1	2
154	MARIAMMAL	29	2	2	1	0	88	48	40	36	1	2
155	SUMITHRA	27	2	1	1	0	90	48	40	36	1	2
156	LAKSHMI	16	1	2	1	1	88					
157	GOPAL	47	4	2	2	0	40	36	30	22	1	2
158	SASIKALA	32	3	2	1	0	36	30	28	22	1	1
159	VIJAYALAKSHMI	14	1	2	1	0	90	46	40	32	1	2
160	PATCHIAPPAN	38	3	2	2	0	88	58	50	40	1	2
161	KOUSALYA	31	3	2	1	0	82	46	43	31	1	2
162	DEVENDRAN	49	4	1	2	0	88	49	41	39	1	2
163	KANNAN	43	4	2	2	0	70	41	37	26	1	1
164	RAMALINGAM	40	4	1	2	0	80	47	40	32	1	2
165	JEYASHANKAR	21	2	2	2	0	78	58	47	32	1	2
166	GAJALAKSHMI	58	5	2	1	0	32	30	27	22	1	1
167	RAMASAMI	40	4	1	2	0	70	55	43	31	1	2
168	KRISNAMOORTHY	49	4	2	2	0	80	44	37	29	1	2
169	THILLAIAMMAL	28	2	2	1	0	78	49	37	27	1	2
170	RAJENDREN	38	3	2	2	0	40	32	20	22	1	2
171	NANDHINI	16	1	2	1	0	86	56	47	32	1	2
172	ROUTRAJ	32	3	2	1	0	70	46	41	28	1	2
173	SATHYA	34	3	2	1	0	38	36	34	30	1	2
174	JEYARAMAN	62	5	3	2	0	82	56	49	37	1	2
175	RAMACHANDRAN	22	2	2	2	0	90	59	44	32	1	2
176	KRISNAMOORTHY	48	4	2	2	0	40	36	32	22	1	2
177	DHANALKSHMI	32	3	2	1	0	90	46	42	36	1	2
178	VIMALA	42	4	2	1	1	90					
179	SAMINATHAN	40	4	1	2	0	76	54	49	32	1	2
180	PARI	32	3	2	2	0	88	48	39	21	1	2
181	VELAYUTHAN	35	3	2	2	0	80	46	40	36	1	2
182	MAALIGA	34	3	2	1	1	90					
183	JEYACHANDRAN	16	1	2	2	0	80	54	49	37	1	2
184	ADAIKALARAJ	24	2	2	2	0	88	48	45	36	1	2
185	MARIAMMAL	17	1	2	1	0	78	46	40	34	1	2
186	RENUKADEVI	20	2	2	1	0	80	47	39	31	1	2
187	SATHYA	18	1	2	1	1	88					
188	RAVI	38	3	1	2	0	90	58	50	42	1	2
189	PALANIAMMAL	37	3	2	1	0	88	48	39	27	1	2
190	LATHA	31	3	2	1	0	80	42	40	27	1	2

191	SARAVANAN 1	14	1	2	2	0	36	30	28	22	1	1
192	BALAMANI	27	2	2	1	0	80	44	40	28	1	2
193	INDRANI	28	2	2	1	0	88	48	36	32	1	2

195	PANNERSELVAM	48	4	1	2	0	90	55	48	37	1	2
196	DHANASELVI	30	3	2	1	0	82	42	40	35	1	2
197	SUDHA	18	1	2	1	0	90	48	40	36	1	2
198	KABILAN	39	3	2	2	0	84	47	38	32	1	2
199	SHEIK	28	2	2	2	0	70	50	40	32	1	2
200	SENNAMA	40	4	3	1	0	38	32	28	22	1	1
201	JEGANNATHAN	35	3	2	2	0	90	57	46	37	1	2
202	TAMILSELVI	36	3	2	1	0	40	36	32	28	1	1
203	PARVATHAM	40	4	2	1	0	86	46	40	32	1	2
204	MUNEESHVARI	35	3	2	1	0	86	46	40	34	1	2
205	MUNUSWAMI	27	2	2	2	0	90	59	47	35	1	2
206	SAMINATHAN	40	4	2	2	0	88	56	47	34	1	2
207	SHANTHI	37	3	1	1	0	38	32	30	24	1	1
208	KOMALA	30	3	1	1	1	40					
209	MURUGESVARI	32	3	2	1	0	80	46	39	32	1	2
210	BALAJI	19	1	2	2	0	78	44	40	32	1	2
211	VENKATESAN	24	2	2	2	0	40	38	33	27	1	1
212	NABISHA	47	4	2	1	0	80	48	39	31	1	2
213	EZHILRANI	28	2	2	1	0	80	56	48	36	1	2
214	KAVITHA	30	3	2	1	0	38	30	28	20	1	1
215	KRISHNAVENI	32	3	2	1	1	90	88				
216	MUTULAXMI	35	3	2	1	1	90	80				
217	VINOTHA	22	2	2	1	1	88	80				
218	RAVIKUMAR	47	4	1	2	0	30	28	26	21	1	1
219	SOUNDARYA	28	2	2	1	0	80	48	42	32	1	2
220	ARJUNAN	16	1	2	2	0	40	36	30	26	1	2
221	DURAISAMI	42	4	2	2	0	88	50	46	31	1	2
222	MALARKODI	31	3	2	1	0	80	42	40	32	1	1
223	KARTHIK	39	3	2	2	0	90	48	36	25	1	2
224	KIRTHIHA	17	1	2	1	0	88	42	40	28	1	2

225	SUDHA	28	2	1	1	1	90	76			
226	VENKATESAN	22	2	2	2	0	78	56	50	35	1
227	PALANI	32	3	2	2	0	40	36	30	32	1
228	PARVATHI	40	4	2	1	0	88	44	40	32	1
229	SELVAMARI	28	2	2	1	0	78	42	40	32	1
230	SUDHA	28	2	2	1	0	40	36	30	22	1
231	SHNTHAKUMARI	40	4	2	1	0	34	32	28	22	1
232	SHANMUGAM	51	5	2	2	0	88				
233	ANJALAIDEVI	26	2	2	1	0	88	50	46	32	1
234	KASTHURI	30	3	2	1	0	78	48	40	32	1
235	KANNIAPPAN	36	3	2	2	0	90	56	50	40	1
236	PALANISAMI	40	4	2	2	0	40	34	32	30	1
237	ABITHA	25	2	1	1	0	90	50	48	32	1
238	GOMATHY	30	3	2	1	0	9048	40	30	26	1
239	JADAMANI	36	3	2	2	0	80	50	44	32	1
240	CHANDRAMATHI	32	3	2	1	0	88	50	47	34	1
241	MUTHU	40	4	2	2	0	80	54	42	36	1
242	SIVAGAMAI	40	4	2	1	0	80	46	42	34	1
243	PRABHU	35	3	2	2	0	90	58	50	32	1
244	SADASIVAM	45	4	1	2	0	78	56	46	32	1
245	EDWARD	58	5	2	2	0	80	48	40	28	1
246	SUGANTHI	17	1	2	1	0	88	48	46	38	1
247	SARALA	23	2	2	1	0	90	42	38	27	1
248	RATHINAVELU	54	5	2	2	0	86	46	40	32	1
249	SARASWATHY 4	23	2	2	1	0	78	46	40	27	1
250	PALANIAMMAL	45	4	2	1	0	70	48	40	34	1
251	SANGEETHA 2	19	1	2	1	0	70	48	40	36	1
252	SNEHA	18	1	2	1	0	90	59	48	31	1
253	SANGEETHA 2	20	2	2	1	0	72	48	40	28	1
254	SUMATHY	35	3	2	1	1	82				
255	SAMUNSEHVARI	15	1	1	1	0	78	48	42	28	1
256	SHANKAR	13	1	1	2	0	80	58	50	45	1
257	MAHESHVARI	35	3	2	1	1	90	80			
258	CHITRA	24	2	2	1	0	90	47	36	28	1
259	KAVITHA	25	2	2	1	0	38	36	32	30	1
260	JANAKI	33	3	2	1	0	80	50	42	34	1
261	LAKSHMI	26	2	2	1	0	40	32	30	22	1
262	MUNUSWAMI	35	3	2	2	0	90	54	48	35	1
263	AYYANAR	35	3	2	2	0	86	54	50	40	1
264	GOPALAKRISHNAN	42	4	1	2	0	86	52	48	32	1
265	VIJAYA	37	3	2	1	0	80	56	48	32	1

* Age Group
1- 10-19 years
2- 20-29 years
3-30-39 years
4- 40-49 years
5-> 50 years

** Sex
1- Female
2-Male

***NYHA
1- Class I
2-Class II
3-Class III